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TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE THIRTY-EIGHTH

PHILADELPHIA
PRINTED FOR THE COLLEGE
1916

NOTICE.

The present volume of TRANSACTIONS contains the papers read before the College from January, 1916, to December, 1916, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WALTER G. ELMER, M.D.

"This volume is published in part by the income from the Francis Houston Wyeth Fund."

DORNAN, PRINTER

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1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES[1]
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS[2]
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
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1907	JAMES TYSON
1910	GEORGE E. DE SCHWEINITZ
1913	JAMES CORNELIUS WILSON
1916	RICHARD H. HARTE

[1] Died four months after his election.
[2] Resigned on account of ill-health.

APR 25 1917
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1916

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Clerk, H. R. M. LANDIS, M.D.

* Resigned.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

DECEMBER, 1916

* Non-resident Fellows.
† Fellows who have commuted dues.

ELECTED

1892. Abbott, Alexander C., M.D., Sc.D. (Hon.), Professor of Hygiene and Bacteriology in the University of Pennsylvania; Member of the Board of Health of Philadelphia. 4229 Baltimore Ave.

1912. Addison, William H. F., A.B., M.B., Assistant Professor of Normal Histology and Embryology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1905. Adler, Lewis H., Jr., M.D., Professor of Diseases of the Rectum in the Philadelphia Polyclinic and College for Graduates in Medicine; formerly Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital. 1610 Arch St.

1914. Aiken, Thomas Gerald, M.D., Assistant Visiting Physician to the Chester County Hospital, West Chester, Pa.; Pathologist to the Country Branch of the Rush Hospital for Tuberculosis. Berwyn, Pa.

1913. Alexander, Emory G., M.D., Surgeon to St. Christopher's Hospital for Children; Associate Surgeon to the Episcopal Hospital; Clinical Professor of Surgery in the Woman's Medical College of Pennsylvania, and Demonstrator of Fracture Dressings in the Jefferson Medical College; Assistant Surgeon to the Kensington Hospital for Women. 1627 Oxford St.

ELECTED

1903. Allen, Alfred Reginald, M.D., Lecturer on Neurological Electrotherapeutics and Instructor in Neurology and Neuropathology in the University of Pennsylvania. 2013 Spruce St.

1906. Allen, Francis Olcott, Jr., A.B., M.D., Surgeon to the Presbyterian Hospital; Assistant Surgeon to the Bryn Mawr Hospital; Surgeon to the Out-patient Department of the Pennsylvania Hospital. 2216 Walnut St.

1896. Allyn, Herman B., M.D., Associate in Medicine in the University of Pennsylvania; Physician to the Philadelphia General Hospital. 501 S. Forty-second St.

1888. Anders, James M., M.D., L.L.D., Professor of Medicine and Clinical Medicine in the Medico-Chirurgical College; Officer de l'Instruction Publique. 1605 Walnut St.

1905. Anspach, Brooke M., M.D., Associate in Gynecology in the University of Pennsylvania; Gynecologist and Obstetrician to the Philadelphia General and the Stetson Hospitals; Assistant Gynecologist to the University Hospital; Assistant Gynecologist to the Bryn Mawr Hospital. 1827 Spruce St.

1905. Appleman, Leighton F., M.D., Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College; Instructor in Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmologist to the Frederick Douglass Memorial Hospital. 308 S. Sixteenth St.

1906. Ashhurst, Astley Paston Cooper, A.B., M.D., Instructor in Surgery in the University of Pennsylvania; Surgeon to the Episcopal Hospital and to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. 811 Spruce St.

1893. Ashton, Thomas G., M.D., Physician to the Philadelphia General Hospital. 1814 S. Rittenhouse Square.

1914. Austin, J. Harold, B.S. (Univ. of Penna.), M.D., Department of Research Med., University of Pennsylvania.

1906. Babbitt, James A., A.B., (Yale), A.M. (Haverford), M.D., Professor of Hygiene and Physical Education at Haverford College; Assistant Laryngologist and Aurist, and Chief of the Out-patient Department for Diseases of the Nose, Throat, and Ear at the German Hospital; Assistant Instructor in Otology in the University of Pennsylvania; Laryngologist to the Out-patient Department of the Children's Hospital. 1901 Chestnut St.

ELECTED

1910. Baer, Benjamin F., Jr., M.D. 2040 Chestnut St.

†1892. Baker, George Fales, B.S., M.D. 1818 Spruce St.

1911. Baldwin, James Harvey, A.B., M.D., Assistant Surgeon to the Methodist Hospital. 1426 Pine St.

1889. Baldy, John Montgomery, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Surgeon to the Gynecean Hospital; Consulting Surgeon to the Jewish and the Frederick Douglass Memorial Hospitals. 2219 De Lancey Place.

1916. Balentine, Percival L., M.D., Assistant Surgeon, Wills Eye Hospital; Demonstrator of Ophthalmic Surgery in the Philadelphia Polyclinic. 2117 Chestnut St.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3709 Powelton Ave.

1911. Barnard, Everett P., M.D., Obstetrician to the Maternity Hospital; Assistant Instructor in Obstetrics in the University of Pennsylvania. 119 S. Nineteenth St.

1883. Baum, Charles, A.M., M.D., Ph.D. 1828 Wallace St.

1908. Beardsley, Edward J. G., M.D., L.R.C.P. (Lond.), Associate Professor of Clinical Medicine in the Jefferson Medical College; Chief Clinical Assistant in the Out-patient Medical Department of the Jefferson Medical College Hospital; Assistant Physician to the Jefferson and Philadelphia Hospitals. 2030 Chestnut St.

*1916. Bell, William Hemphill, M.D., Surgeon U. S. Navy. U. S. S. Wyoming, Navy Yard, New York.

*1874. Bennett, W. H., A.M., M.D., Physician-in-Charge of the Seashore House for Invalid Children, and of the Seaside House for Invalid Women, Atlantic City; formerly Physician to the Episcopal Hospital, and Physician-in-Charge of St. Christopher's Hospital for Children. Children's Seashore Home, Atlantic City, N. J.

1896. Beyea, Henry D., M.D., Associate in Gynecology and Assistant Demonstrator of Obstetrics in the University of Pennsylvania; Assistant Surgeon to the Gynecean Hospital. 1734 Spruce St.

*1903. Biggs, Montgomery H., M.D., Surgeon to the Rutherford Hospital; Chief Surgeon of the Carolina, Churchfield and Ohio Railway. Rutherfordton, N. C.

1908. Bland, Pascal Brooke, M.D., Chief Clinical Assistant in the Gynecological Department of the Jefferson Medical College Hospital; Instructor in Gynecology in the Jeffer-

ELECTED

son Medical College; Gynecologist to St. Joseph's Hospital; Assistant Gynecologist to the Philadelphia General Hospital. 1621 Spruce St.

1894. Bochroch, Max H., M.D., Demonstrator of Neurology and Chief Clinical Assistant in the Nervous Department of the Jefferson Medical College Hospital; Neurologist to the Out-patient Department of St. Joseph's Hospital. 1539 Pine St.

1896. Boger, John A., A.M., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Stetson Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 2213 N. Broad St.

1910. Boice, J. Morton, A.B., M.D., Gynecologist to the Out-patient Department of St. Joseph's Hospital; Lecturer on Chemistry to the Training School of St. Joseph's Hospital; Obstetric Registrar to the Philadelphia General Hospital. 4020 Spruce St.

1911. Bonney, Charles W., A.B., M.D., Demonstrator of Applied Anatomy in the Jefferson Medical College; Assistant Surgeon, Department of Oral Surgery, Philadelphia General Hospital. 1117 Spruce St.

1913. Boston, L. Napoleon, A.M., M.D., Professor of Physical Diagnosis in the Medico-Chirurgical College; Physician to the Philadelphia General Hospital; Pathologist to the Frankford Hospital. 1819 Chestnut St.

1891. Boyd, George M., M.D., Professor of Obstetrics in the Medico-Chirurgical College; Obstetrician to the Medico-Chirurgical Hospital; Physician to the Philadelphia Lying-in Charity; Obstetrician and Gynecologist to the Philadelphia General Hospital. 1909 Spruce St.

1907. Boyer, Henry Percival, M.D., Physician to the Stetson Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Chief Medical Inspector Pennsylvania Department of Health. 4602 Baltimore Ave.

1907. Bradley, William N., Ph.G., M.D., Pediatrist to Howard Hospital. 1638 S. Broad St.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1891. Brinton, Lewis, M.D., Physician-in-Chief to the American Hospital for Diseases of the Stomach. 1933 Spruce St.

1900. Brinton, Ward, A.M., M.D., Visiting Physician to the Tuberculosis Department of the Philadelphia General

ELECTED

Hospital; Visiting Physician to the Hospital for Poor Consumptives at White Haven, Pa.; Visiting Physician to the Philadelphia Jewish Sanatorium for Consumptives. 1423 Spruce St.

1907. Brooks, Macy, A.B. (Princeton), M.D., Assistant Genito-urinary Surgeon to the Philadelphia General Hospital. 1321 Spruce St.

1916. Brown, Samuel Horton, M.D. 1901 Mt. Vernon St.

1887. Brubaker, Albert P., A.M., M.D., Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College; Professor of Physiology and Hygiene in the Drexel Institute of Science, Art, and Industry. 3426 Powelton Ave.

1916. Buckley, Albert Coulson, M.D., Clinical Director, Friends' Hospital, Frankford; Acting Clinical Professor of Mental Diseases, University of Pennsylvania; Visiting Physician to the Psychopathic Wards, Philadelphia General Hospital; Alienist to the Orthopædic Hospital and Infirmary for Nervous Diseases. Friends' Hospital, Frankford, Phila.

1906. Burns, Stillwell C., M.D., Lecturer on Surgery in the Medico-Chirurgical College; Assistant Surgeon to the Medico-Chirurgical Hospital. 1326 Spring Garden St.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 1918 Spruce St.

1906. Butler, Ralph, M.D., Professor of Diseases of the Nose and Throat in the Philadelphia Polyclinic and the College for Graduates in Medicine; Chief of the Dispensary for Diseases of the Ear at the University Hospital; Instructor in Otology in the University of Pennsylvania; Laryngologist and Aurist to the German Hospital. 1926 Chestnut St.

*1912. Butt, William Redfield, M.D. 222 Cleveland Ave., N. W. Canton, Ohio.

*1908. Cadbury, William W., A.M., M.D., Canton Christian College, Canton, China.

†1907. Cadwalader, Williams B., M.D., Pathologist and Clinical Assistant to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Instructor in Nervous Diseases in the Philadelphia Polyclinic. 1710 Locust St.

1905. Cameron, George A., M.D., Physician to the Germantown Hospital. S. E. cor. Schoolhouse Lane and Greene St., Germantown.

ELECTED

1905. Carmany, Harry S., M.D., Surgeon to St. Timothy's Hospital, Roxborough; Surgeon to the Dispensary of the Episcopal Hospital. 366 Green Lane, Roxborough.

1910. Carnett, John Berton, M.D., Associate in Surgery in the University of Pennsylvania; Assistant Surgeon to the University and the Philadelphia General Hospitals; Surgeon to the Chestnut Hill Hospital; Consulting Surgeon to the Henry Phipps Institute, to the Phœnixville Hospital, and to the Eastern Pennsylvania Institution for the Feeble-minded and Epileptic. 123 S. Twentieth St.

1905. Carpenter, Herbert B., M.D., Physician to the Dispensary of the Children's Hospital. 1805 Spruce St.

1895. Carpenter, John T., M.D., Lecturer on Ophthalmology in the University of Pennsylvania; Assistant Ophthalmic Surgeon to the University Hospital; Attending Ophthalmologist to the Bryn Mawr Hospital. 2039 Chestnut St.

1892. Cattell, Henry W., A.M., M.D., Editor of *Medical Notes and Queries*. 3709 Spruce St.

1900. Chance, Burton, M.D., Attending Surgeon to the Wills Eye Hospital; Consulting Ophthalmologist Eastern Pennsylvania Institution for the Feeble-minded and Epileptic; Ophthalmic Surgeon to the Pennsylvania Railroad Company. 235 S. Thirteenth St.

*1868. Cheston, D. Murray, M.D. Harwood P. O., Md.

1897. Cheston, Radcliffe, M.D., Visiting Physician to the Chestnut Hill Hospital; Consulting Physician to the Germantown Hospital, and to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill.

1904. Christian, Hilary M., M.D., Clinical Professor of Genito-urinary Diseases in the Medico-Chirurgical College. 1321 Spruce St.

1903. Chrystie, Walter, M.D. Bryn Mawr, Pa.

1899. Clark, John G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital. 2017 Walnut St.

1896. Cleveland, Arthur H., M.D., Clinical Professor of Laryngology in the Medico-Chirurgical College; Laryngologist to the Medico-Chirurgical Hospital; Laryngologist and Aurist to the Presbyterian Hospital, and to Pennsylvania Institution for Deaf and Dumb. 256 S. Fifteenth St.

1910. Cloud, J. Howard, M.D., Assistant Physician to the Bryn Mawr Hospital; Attending Physician to the Children's House of Bryn Mawr Hospital. 7 W. Lancaster Ave., Ardmore, Pa.

ELECTED

1903. Coates, George Morrison, A.B., M.D., Surgeon to the Out-Patient Department for Diseases of the Ear, Throat, and Nose of the Pennyslvania Hospital; Professor of Diseases of the Ear in the Philadelphia Polyclinic; Laryngologist to the Tuberculosis Department of the Philadelphia General Hospital; Consulting Laryngologist to the Philadelphia Orphanage. 1736 Pine St.

1908. Codman, Charles A. E., M.D., Physician to the American Oncologic Hospital. 4116 Spruce St.

1907. Cohen, Myer Solis, A.B., M.D., Pediatrist to the Jewish Hospital, and to the Philadelphia Jewish Sanatorium for Consumptives, Eagleville, Pa.; Assistant Visiting Physician to the Philadelphia General Hospital; Consulting Physician to the Home for Consumptives, Chestnut Hill. 4102 Girard Ave.

1888. Cohen, Solomon Solis, M.D., Professor of Clinical Medicine in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital, to the Philadelphia General Hospital, to the Jewish Hospital, and to the Rush Hospital. 1525 Walnut St.

1898. Coles, Stricker, M.D., Assistant Professor of Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson and the Philadelphia General Hospitals; Visiting Physician to the Philadelphia Lying-in-Charity Hospital. 2103 Walnut St.

1901. Coley, Thomas Luther, A.B., M.D., Attending Physician Methodist Episcopal Hospital, Philadelphia. 256 S. Fifteenth St.

1903. Coplin, W. M. L., M.D., Professor of Pathology in the Jefferson Medical College; Pathologist to and Director of the Laboratories of the Jefferson Medical College Hospital; Pathologist to the Philadelphia General Hospital and to the Friends' Asylum for the Insane, Frankford; Bacteriologist to the Pennsylvania State Board of Health. 606 S. Forty-eighth St.

1912. Copp, Owen, A.B., M.D., Physician-in-chief and Superintendent of the Pennsylvania Hospital for the Insane. Pennsylvania Hospital for the Insane, Forty-fourth and Market Sts.

1911. Cornell, Walter Stewart, A.B., M.D., Director of Medical Inspection of Public Schools of the City of Philadelphia; Chief of Medical Staff of the House of Detention; Lecturer on Osteology in the University of Pennsylvania; Lieutenant, Medical Reserve Corps, U. S. A. 729 City Hall.

ELECTED

1914. Corson, Edward Foulke, M.D., Physician for Diseases of the Skin, Presbyterian Hospital Dispensary; Assistant Dermatologist, Children's Hospital Dispensary. Cynwyd, Pa.

1907. Council, Malcolm S., M.D., Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Cathcart Home at Devon. Bryn Mawr, Pa.

*1909. Craig, Alexander R., A.M., M.D. 535 N. Dearborn St., Chicago, Ill.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute, University of Pennsylvania; Visiting Physician to the White Haven Sanatorium; Physician-in-Charge of the Tuberculosis Class of the Presbyterian Hospital. 244 S. Twenty-first St.

1907. Crampton, George S., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital and the Philadelphia Hospital for Contagious Diseases; Assistant Surgeon to the Wills Hospital; Lecturer on Physiologic Optics in the Philadelphia Polyclinic and School for Graduates in Medicine; Ophthalmologist to the Philadelphia Orphanage. 1700 Walnut St.

1904. Cruice, John M., M.D., Physician to the Henry Phipps Institute of the University of Pennsylvania; Physician to the Medical Dispensary of St. Agnes' Hospital; Instructor in Medicine in the University of Pennsylvania. 1932 Spruce St.

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Cal.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1903. Da Costa, John C., Jr., M.D., Associate Professor of Medicine in the Jefferson Medical College; Assistant Physician to the Jefferson Medical College Hospital; Hematologist to the German Hospital; Consulting Physician to the Northwestern General Hospital. 264 S. Fifteenth St.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospitals. 2045 Walnut St.

1887. Daland, Judson, M.D., Professor of Clinical Medicine in the Graduate School of Medicine, University of Pennsylvania. 317 S. Eighteenth St.

ELECTED

°1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital. 5923 Greene St., Germantown.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1888. Davis, Edward P., A.M., M.D., Professor of Obstetrics in the Jefferson Medical College and in the Philadelphia Polyclinic; Visiting Obstetrician to the Jefferson and the Polyclinic Hospitals; Obstetrician and Gynecologist to the Philadelphia General Hospital. 250 S. Twenty-first St.

1889. Davis, Gwilym G., M.D. (Univ. of Penna. and Göttingen), LL.D., M.R.C.S. (Eng.), Professor of Orthopedic Surgery in the University of Pennsylvania; Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; Orthopedic Surgeon to the Philadelphia General Hospital; Consulting Surgeon to St. Joseph's Hospital. 1814 Spruce St.

1916. Davis, Warren B., B.D., Oral Surgeon, Philadelphia General Hospital; Assistant Rhinologist and Otologist St. Agnes' Hospital; Assistant Demonstrator of Anatomy Jefferson Medical College; Clinical Assistant in Surgical Dispensary Jefferson Hospital. 135 S. Eighteenth St.

1900. Davisson, Alex. Heron, M.D. 1017 S. Forty-sixth St.

1894. Deaver, Harry C., M.D., Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Episcopal Hospital, and to the Children's Hospital of the Mary J. Drexel Home; Surgeon-in-Chief to the Kensington Hospital for Women. 1415 N. Broad St.

1887. Deaver, John B., M.D., D.Sc., LL.D., Professor of the Practice of Surgery in the University of Pennsylvania; Surgeon-in-Chief to the German Hospital. 1634 Walnut St.

1902. Dehoney, Howard, M.D. 240 S. Thirteenth St.

1885. Dercum, Francis X., A.M., M.D., Ph.D., Professor of Nervous and Mental Diseases in the Jefferson Medical College; Consulting Neurologist to the Philadelphia General Hospital; Foreign Corresponding Member of the Neurological Society of Paris, and Corresponding Member of the Psychiatric and Neurological Society of Vienna. 1719 Walnut St.

1908. Despard, Duncan L., M.D., Surgeon to the Abington Memorial Hospital; Assistant Surgeon to the Jefferson Medical College Hospital; Demonstrator of Clinical Surgery in the Jefferson Medical College; Associate in Gynæcology in the Philadelphia Polyclinic Hospital. 1806 Pine St.

ELECTED

1912. Dewey, J. Hiland, Ph.B., M.D., Assistant Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to St. Francis' Hospital, Trenton, N. J. 1436 Diamond St.

*1911. Dickson, Frank D., M.D. St. Regis Hotel, Kansas City, Mo.

1908. Dillard, Henry K., Jr., M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Physician to the Dispensary of the Mary J. Drexel Home. 234 S. Twentieth St.

1891. Dixon, Samuel G., M.D., LL.D., Commissioner of Health of Pennsylvania; President of the Academy of Natural Sciences of Philadelphia; Member of the Philadelphia Bar. Bryn Mawr, Pa.

*1897. Dorland, W. A. Newman, A.M., M.D., Professor of Gynecology in the Post-graduate Medical School of Chicago; Professor of Obstetrics in the Chicago College of Medicine and Surgery; Visiting Obstetrician to Cook County Hospital; First Lieutenant, Medical Reserve Corps, U. S. Army. 7 West Madison St., Chicago, Ill.

1907. Dorrance, George Morris, M.D., Surgeon to St. Agnes' Hospital; Demonstrator of Applied Anatomy in the Dental Department of the University of Pennsylvania. 2025 Walnut St.

°1864. Downs, R. N., M.D., Consulting Physician to the Germantown Hospital. 5916 Greene St., Germantown.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St., Germantown.

1910. Drayton, William, Jr., M.D., Physician to the Philadelphia Hospital for Contagious Diseases; Physician to the Out-patient Department of the Pennsylvania Hospital; Physician to the Pennsylvania Institute for the Instruction of the Blind; Assistant Physician to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. 1316 Locust Street.

1881. Dulles, Charles Winslow, M.D., Consulting Surgeon of the Rush Hospital. 4101 Walnut St.

1911. Earnshaw, Henry Culp, M.S., M.D., Attending Physician to the Hospital of the Good Shepherd, Rosemont; Assistant Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Bryn Mawr Children's Hospital; Pennsylvania Railroad Surgeon. Bryn Mawr, Pa.

ELECTED

*1887. Edwards, William A., M.D., Professor of Pediatrics in the Medical Department of the University of California. Fifth and Spring Sts., Los Angeles, Cal.

1911. Eliason, Eldridge E., B.A., M.D., Assistant Instructor in Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Assistant Surgeon to the Howard Hospital; Surgeon to the Out-patient Department of the Children's Hospital. 320 S. Sixteenth St.

1904. Elmer, Walter G., B.S., M.D., Instructor in Orthopedic Surgery in the University of Pennsylvania; Assistant Orthopedic Surgeon to the University Hospital; Orthopedic Surgeon to the Jewish Hospital; Surgeon to the Pennsylvania Training School for Children. 1801 Pine St.

1896. Ely, Thomas C., A.M., M.D. 2041 Green St.

1901. Erck, Theodore A., M.D., Associate in Gynecology in the Philadelphia Polyclinic and College for Graduates in Medicine; Associate Surgeon to the Gynecean Hospital. 251 S. Thirteenth St.

1893. Eshner, Augustus A., M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic and College for Graduates in Medicine; Physician to the Philadelphia General Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Physician to Mercy Hospital. 1019 Spruce St.

*1905. Evans, Joseph S., Jr., A.B., M.D., Professor of Clinical Medicine in the University of Wisconsin; Consulting Physician, Madison General Hospital. University of Wisconsin, Madison, Wis.

1905. Evans, William, M.D. 4007 Chestnut St.

1912. Eves, Curtis C., M.D., Aural and Laryngeal Surgeon to the Episcopal Hospital; Assistant in the Out-patient Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Demonstrator of Operative Surgery of the Ear, Nose, and Throat in the Philadelphia Polyclinic. 1700 Walnut St.

1894. Faries, Randolph, M.D. 2007 Walnut St.

†1903. Farr, Clifford B., A.B., M.D., Professor of Diseases of the Stomach, etc., in the Philadelphia Polyclinic; Associate in Medicine in the University of Pennsylvania; Assistant Physician to the Philadelphia General Hospital. 117 S. Twenty-second Street.

ELECTED

1893. Farr, William W., M.D., Physician to the Leamy Home. Springfield Ave. and Lincoln Drive, Chestnut Hill.

1884. Fenton, Thomas H., M.D., Ophthalmologist to St. Vincent's Home, to the Home for Aged Couples, to the Baptist Home, and to the House of the Good Shepherd. 1319 Spruce St.

1907. Ferguson, Albert D., M.D., Physician-in-charge of the Widener Memorial School for Crippled Children. 50 E. Johnson St., Germantown.

1907. Fetterolf, George, A.B., M.D., Sc.D., Laryngologist to the Henry Phipps Institute for Tuberculosis; Laryngologist to the White Haven Sanatorium; Consulting Laryngologist to the Phœnixville Hospital; Demonstrator of Anatomy in the University of Pennsylvania. 134 S. Twentieth St.

1907. Fife, Charles A., A.B., M.D., Instructor in Pediatrics in the University of Pennsylvania; Pediatrist to the Presbyterian Hospital; Physician to the St. Christopher's Hospital for Children; Assistant Physician to the Philadelphia General Hospital. 2033 Locust St.

1884. Fisher, Henry M., M.D. 1027 Pine St.

1910. Fisher, John Monroe, M.D., Associate Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Philadelphia, St. Agnes', and Phœnixville Hospitals; Assistant Gynecologist to the Jefferson Medical College Hospital. 222 S. Fifteenth St.

1888. Flick, Lawrence F., M.D. 736 Pine St.

1916. Forst, John R., M.D. 166 W. Coulter St., Germantown.

1908. Foulkrod, Collin, M.D., Obstetrician to the Maternity House of the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in Jefferson Medical College; Gynecologist to the Dispensary of the Presbyterian Hospital. 4005 Chestnut St.

1908. Fox, Herbert, M.D., Director of the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Pathologist to the Rush Hospital; Pathologist to the Children's Hospital. 3902 Locust St.

†1885. Fox, Joseph M., M.D. Torresdale, Pa.

1906. Fraley, Frederick, Jr., A.B., M.D. 1701 De Lancey Place.

ELECTED

1903. Francine, Albert Philip, A.M., M.D., Associate in Medicine in the University of Pennsylvania; Visiting Physician to the Philadelphia General Hospital, Department of Tuberculosis; Physician-in-Chief to the State Dispensary for Tuberculosis, Philadelphia. 264 S. Twenty-first St.

1897. Frazier, Charles H., A.B., M.D., Sc.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University Hospital. 1724 Spruce St.

†1890. Freeman, Walter J., M.D., Emeritus Professor of Laryngology in the Philadelphia Polyclinic; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb. 1832 Spruce St.

1916. Funk, Elmer Hendricks, M.D. 1318 Spruce St.

1910. Furbush, Charles Lincoln, M.D. 1501 Spruce St.

1889. Fussell, M. Howard, M.D., Professor of Applied Therapeutics in the University of Pennsylvania; Physician to the University Hospital, the Episcopal Hospital, St. Timothy's Hospital, and St. Mary's Hospital. 2035 Walnut St.; 421 Lyceum Ave., Roxborough.

1899. Gamble, Robert G., M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1912. Gaskill, Henry Kennedy, M.D., Assistant Professor of Dermatology in the Jefferson Medical College; Attending Dermatologist to the Philadelphia General Hospital. 1610 Spruce St.

1873. Gerhard, George S., M.D., Physician-in-Chief to the Bryn Mawr Hospital; Consulting Physician to Bryn Mawr College; Consulting Physician to Villa Nova College. Fifty-eighth Street and Overbrook Ave.

1902. Ghriskey, Albert A., M.D. 3936 Walnut St.

1899. Gibbon, John H., M.D., Professor of Surgery in the Jefferson Medical College; Surgeon to the Pennsylvania and the Bryn Mawr Hospitals. 1608 Spruce St.

1908. Gildersleeve, Nathaniel, M.D., Professor of Microbiology and Bacteriopathology; The Thomas W. Evans Museum and Dental Institute School of Dentistry University of Pennsylvania. School of Dentistry, University of Pennsylvania.

1913. Ginsburg, Nathaniel, M.D., Surgeon to the Jewish Hospital; Associate in Surgery in the Philadelphia Polyclinic and College for graduates in Medicine; Assistant Surgeon to Mt. Sinai Hospital; Instructor in Anatomy in the University of Pennsylvania. 1704 Pine St.

ELECTED

1897. Girvin, John H., M.D., Physician for Diseases of Women at the Presbyterian Hospital; Instructor in Obstetrics in the University of Pennsylvania. 2120 Walnut St.

1906. Gittings, J. Claxton, M.D., Instructor in Pediatrics in the University of Pennsylvania; Assistant Pediatric Physician to the University Hospital; Visiting Physician to the Children's Hospital; Consulting Physician to the Sheltering Arms and the Presbyterian Orphanage. 3903 Chestnut St.

1905. Given, Ellis E. W., M.D., Surgeon to the Philadelphia Freemasons Memorial Hospital of the Masonic Home, Elizabethtown, Pa.; Surgeon to the Dispensary of the Episcopal Hospital. 2018 Chestnut St.

1894. Gleason, E. B., S.B., M.D., LL.D., Professor of Otology in the Medico-Chirurgical College. 2033 Chestnut St.

1906. Goepp, R. Max, M.D., Professor of Clinical Medicine, Dean of the College Department, and Secretary of the Faculty of the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Professor of Clinical Medicine in the Jefferson Medical College; Assistant Visiting Physician to the Philadelphia General Hospital. 124 S. Eighteenth St.

1906. Goldberg, Harold G., M.D., Ophthalmic Surgeon to the Episcopal Hospital and to the Kensington Hospital for Women; First Lieutenant, Medical Reserve Corps, U. S. Army. 1925 Chestnut St.

1893. Goodell, W. Constantine, M.D. 300 S. Thirteenth St.

1908. Goodman, Edward H., M.D., Associate in Medicine in the University of Pennsylvania; Consultant to the Medical Dispensary of the University Hospital; Assistant Physician to the University Hospital; Assistant Physician to the Philadelphia General Hospital. 248 S. Twenty-first St.

1905. Gordon, Alfred, M.D., Neurologist to the Mt. Sinai, the Northwestern General, and the Douglass Memorial Hospitals. 1812 Spruce St.

†1897. Gould, George M., A.M., M.D. 215 Atlantic Ave., Atlantic City, N. J.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and the Philadelphia General Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1885. Graham, John, M.D. 326 S. Fifteenth St.

ELECTED

1904. Grayson, Charles P., M.D., Professor of Laryngology and Rhinology in the University of Pennsylvania; Physician-in-Charge of the Throat and Nose Department of the University Hospital; Otolaryngologist to the Philadelphia General Hospital. 262 S. Fifteenth St.

1910. Greenman, Milton J., M.D., Sc.D., Director of the Wistar Institute of Anatomy and Biology. Wistar Institute of Anatomy and Biology, Thirty-sixth St. and Woodland Ave.

1883. Griffith, J. P. Crozer, M.D., Professor of Pediatrics in the University of Pennsylvania; Corresponding Member of the Société de Pédiatrie de Paris. 1810 Spruce St.

1912. Griscom, J. Milton, B.S., M.D., Assistant Surgeon to the Wills Eye Hospital; Chief of Clinic of the Eye Dispensary of the Presbyterian Hospital. 1925 Chestnut St.

1911. Gummey, Frank Bird, M.D., Visiting Physician to the Germantown Hospital and Dispensary; Visiting Physician to the Midnight Mission. 5418 Greene St., Germantown.

*1902. Gwyn, Norman B., M.D., Instructor in Medicine in the University of Pennsylvania. 20 S. Twenty-first St.

1894. Hamill, Samuel McC., M.D., Professor of Diseases of Children in the Philadelphia Polyclinic and College for Graduates in Medicine; Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. Hand, Alfred, Jr., M.D., Visiting Physician to the Children's Hospital, to the Children's Hospital of the Mary J. Drexel Home, and to the Methodist Hospital. 1724 Pine St.

1886. Hansell, Howard F., M.D., Professor of Ophthalmology in the Jefferson Medical College; Ophthalmic Surgeon to the Philadelphia General Hospital and to the Jefferson Medical College Hospital; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. N. E. Cor. 17th and Walnut Sts.

1889. Hare, Hobart A., M.D., Professor of Therapeutics, Materia Medica, and Diagnosis in the Jefferson Medical College, 1801 Spruce St.

1903. Hart, Charles D., A.M., M.D., Inspector and Secretary of the Eastern State Penitentiary; National Executive Committee and Chairman of the Philadelphia Committee, Boy Scouts of America. 1317 Walnut St.

ELECTED

1885. Harte, Richard H., M.D., Adjunct Professor of Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania and the Orthopædic Hospitals; Consulting Surgeon to St. Mary's, St. Timothy's, and the Bryn Mawr Hospitals. 1503 Spruce St.

1888. Hartzell, Milton B., A.M., M.D., LL.D., Professor of Dermatology in the University of Pennsylvania. 3644 Chestnut St.

1907. Hatfield, Charles James, A.B. (Princeton), M.D., Executive Director of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. 2008 Walnut Street.

1872. Hays, I. Minis, M.D. 266 S. Twenty-first St.

1911. Heed, Charles R., M.D., Associate Professor of Ophthalmology in the Philadelphia Polyclinic; Instructor in Ophthalmology in the Jefferson Medical College; Ophthalmologist to Girard College. 1402 Spruce St.

1908. Heineberg, Alfred, M.D., Associate in Gynecology in the Jefferson Medical College; Assistant Gynecologist to St. Agnes' and to Mt. Sinai Hospitals. 1642 Pine St.

1901. Heisler, John C., M.D., Professor of Anatomy in the Medico-Chirurgical College. 3829 Walnut St.

1884. Henry, Frederick P., A.M., M.D., Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania; Physician to the Philadelphia General Hospital. 114 S. Eighteenth St.

1903. Henry, J. Norman, M.D., Physician to the Pennsylvania Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Assistant Physician to the Philadelphia General Hospital. 1906 Spruce St.

1891. Hewson, Addinell, A.B., A.M., M.D., Professor of Anatomy in the Philadelphia Polyclinic and College for Graduates in Medicine; Professor of Anatomy and Histology in the Temple University; Surgeon to St. Timothy's Hospital, Roxborough. 2120 Spruce St.

1909. Higbee, William S., M.D., President of the Pennsylvania State Board of Examiners for Registration of Nurses. 1703 S. Broad St.

1910. Hill, Howard Kennedy, M.D., Assistant Instructor in Medicine in the University of Pennsylvania; Physician to the Children's Medical Dispensary of the Presbyterian

ELECTED

Hospital; Visiting Physician to the University Settlement, and to the Day Nursery; Assistant Physician to the Medical Dispensary of the Children's Hospital. 314 S. Seventeenth Street.

1897. Hinkle, William M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory. 1323 N. Thirteenth St.

*1892. Hinsdale, Guy, A.M., M.D., Associate Professor of Climatology in the University of Pennsylvania. Hot Springs, Virginia.

1888. Hirsh, A. Bern, M.D., Physician to the Home for Aged Couples. 22 S. Twenty-first St.

1888. Hirst, Barton Cooke, A.B., M.D., LL.D., Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Philadelphia General and the Howard Hospitals. 1821 Spruce Street.

1903. Hirst, John Cooke, M.D., Associate in Obstetrics, University of Pennsylvania; Gynecologist and Obstetrician to the Philadelphia General Hospital; Obstetrician to St. Agnes' Hospital; Gynecologist to the American Hospital for Diseases of the Stomach; Assistant Obstetrician to the University Hospital. 1823 Pine St.

1908. Hitchens, Arthur Parker, M.D. Glenolden, Pa.

1905. Hodge, Edward Blanchard, A.B., M.D., Surgeon to the Presbyterian and the Children's Hospitals; Surgeon to the Out-patient Department of the Pennsylvania Hospital; Associate Surgeon to the Widener Memorial School. 346 S. Sixteenth Street.

1913. Hoffman, Clarence, M.D., 425 S. Carlisle St.

°1885. Holland, James W., A.M., M.D., Sc.D., Emeritus Professor of Medical Chemistry and Toxicology and Dean of the Jefferson Medical College. 2006 Chestnut St.

1906. Holloway, Thomas B., M.D., Professor of Ophthalmology in the Philadelphia Polyclinic and School for Graduates in Medicine; Instructor in Ophthalmology in the University of Pennsylvania; Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases; Ophthalmologist to the Pennsylvania Institution for the Instruction of the Blind at Overbrook. 1819 Chestnut St.

1914. Hooker, Richard S., M.D. 110 S. Nineteenth St.

1908. Hoyt, Daniel M., M.D., Assistant Visiting Physician to the Philadelphia General Hospital. 3604 Chestnut St.

ELECTED

*1912. Huber, G. Carl, M.D., Professor of Anatomy and Director of the Anatomic Laboratories in the University of Michigan. 1330 Hill St., Ann Arbor, Mich.

1892. Hughes, William E., M.D., Visiting Physician to the Philadelphia General Hospital; Pathologist to the Presbyterian Hospital. 3945 Chestnut St.

1912. Hunter, John W., B.S., M.D. 2042 Pine St.

1898. Hutchinson, James P., M.D., Surgeon to the Pennsylvania, the Methodist, the Children's, St. Timothy's, and the Bryn Mawr Hospitals; Adjunct Professor of Surgery in the University of Pennsylvania. 133 S. Twenty-second St.

1871. Ingham, James V., M.D. 1811 Walnut St.

*1885. Jackson, Edward, A.M., M.D., Sc.D., Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. 318 Majestic Building, Denver, Col.

*1906. Jacobs, Francis Brinton, B.S., M.D., Assistant Surgeon to the Chester County Hospital. Whitford, Pa.

1913. Jefferys, William Hamilton, A.B., A.M., M.D., Surgeon to St. Luke's Hospital, Shanghai; Professor of Surgery in St. John's University, Shanghai; Editor of the China Medical Journal. 100 W. Walnut Lane, Germantown.

1898. Johnson, Russell H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill, Philadelphia.

1900. Jones, Charles James, A.M., M.D., LL.D., Ophthalmic Surgeon to St. Joseph's Hospital; Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Consulting Ophthalmologist to St. Vincent's Home. 1507 Locust Street.

1914. Jones, Isaac H., A.B., A.M., M.D. 1831 Chestnut St.

1913. Jones, John F. X., B.S., A.B., A.M., M.D., Surgeon to St. Joseph's Hospital. 1815 Spruce St.

1900. Jopson, John H., M.D., Professor of Surgery in the Philadelphia Polyclinic; Associate in Surgery in the University of Pennsylvania; Surgeon to the Presbyterian and the Children's Hospitals. 1824 Pine St.

1900. Judson, Charles F., A.B., M.D., Physician to St. Christopher's Hospital for Children, to the Southern Home for Destitute Children and to the Sheltering Arms. 1005 Spruce St.

ELECTED

1902. Jump, Henry D., M.D., Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Medical Dispensary of the University Hospital. 4634 Chester Ave.

1886. Jurist, Louis, M.D. 916 N. Broad St.

1903. Kalteyer, Frederick J., M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College; Chief of the Out-patient Department, Assistant Attending Physician, and Hematologist to the Jefferson Medical College Hospital; Pathologist to Philadelphia Lying-in Charity. 1533 Pine St.

*1910. Karsner, Howard T., M.D., Professor of Pathology in the Western Reserve University Medical School. Lakeside Hospital, Cleveland, Ohio.

†1867. Keen, William W., M.D., LL.D., (Hon.) F.R.C.S. (Eng. and Edin.), Emeritus Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Membre Correspondant Etranger de la Société de Chirurgie de Paris; Honorary Member of the Société Belge de Chirurgie and of the Clinical Society of London; Ehren mitglied der deutschen Gesellschaft für Chirurgie. 1729 Chestnut St.

1912. Keene, Floyd E., M.D., Instructor in Gynecology in the University of Pennsylvania; Assistant Gynecologist to the University Hospital; Gynecologist to the Chestnut Hill Hospital. 116 S. Nineteenth St

1913. Kelly, Francis Joseph, M.D. 1809 Chestnut St.

*1887. Kelly, Howard A., A.B., M.D., LL.D. (Aberdeen, Wash. and Lee, and Univ. of Pa.), Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.; Hon. Fellow of the Edinburgh Obstetrical Society, the Royal Academy of Medicine of Ireland, and of the Glasgow Obstetrical and Gynecological Society. 1418 Eutaw Place, Baltimore, Md.

1909. Kelly, James A., A.M., M.D., Visiting Surgeon to St. Mary's and St. Timothy's Hospitals; Associate in Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Visiting Surgeon to St. Joseph's Hospital. 1510 N. Seventeenth St.

1912. Kelly, Thomas C., A.M., M.D., Assistant Instructor of Medicine in the University of Pennsylvania; Pediatrician to St. Mary's Hospital; Physician to Out-patient Department of Germantown Hospital. 105 School Lane, Germantown.

ELECTED

1898. Kempton, Augustus F., M.D. 2118 Pine St.

1905. Kercher, Delno E., M.D. 1534 Pine St.

1913. Klopp, Edward J., M.D., Instructor in Surgery in the Jefferson Medical College; Assistant Surgeon to the Germantown Hospital; Chief Clinical Assistant in the Surgical Department of the Jefferson Hospital; Assistant Surgeon to the Out-patient Department of the Pennsylvania Hospital. 1223 Spruce St.

1895. Kneass, Samuel S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania. 1510 Walnut St.

1908. Knipe, Jay C., M.D., Ophthalmologist to the Jewish Hospital; Assistant Ophthalmologist to the Philadelphia General Hospital, and to the Mary J. Drexel Home; Chief of the Eye Clinic at the Jefferson Medical College Hospital; Demonstrator of Osteology and Syndesmology in the Jefferson Medical College. 2035 Chestnut St.

1908. Knowles, Frank Crozer, M.D., Instructor in Dermatology in the University of Pennsylvania; Clinical Professor of Dermatology in the Woman's Medical College; Dermatologist to the Presbyterian Hospital; Assistant Dermatologist to the Dispensary of the Pennsylvania Hospital. 2022 Spruce Street.

1914. Kolmer, John A., M.D., Dr. P.H., M.Sc., Assistant Professor of Experimental Pathology in the University of Pennsylvania; Professor of Pathology and Pathologist to the Department of Dermatological Research, Philadelphia Polyclinic; Pathologist to the Philadelphia Hospital for Contagious Diseases; Serologist to St. Agnes' and St. Timothy's Hospitals. 927 S. St. Bernard St.

1904. Krauss, Frederick, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic and Aural Surgeon to St. Christopher's Hospital for Children and Dispensary; Ear, Nose, and Throat Physician to the Children's Seashore House for Invalid Children, Atlantic City, N. J. 1701 Chestnut Street.

1905. Kremer, Walter H., M.D., 5904 Greene St., Germantown.

1914. Krumbhaar, Edward B., A.B., Ph.D., M.D., Assistant Professor in Research Medicine University of Pennsylvania; Physician to Out-patient Department, Pennsylvania Hospital. W. Mermaid Lane, Chestnut Hill.

ELECTED

1900. Krusen, Wilmer, M.D., Professor of Gynecology in the Medical Department of Temple University; Chief Gynecologist to the Samaritan and the Garretson Hospitals; Consulting Gynecologist to the Charity and Mercy Hospitals. 127 N. Twentieth St.

1913. Laird, John Lohra, A.B., M.D., Assistant Surgeon in Genito-Urinary Diseases to the University Hospital; Associate in Serology in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania; Assistant Instructor in Genito-Urinary Surgery in the University of Pennsylvania. 247 S. Seventeenth St.

1909. Laird, J. Packard, M.D., Visiting Physician to the Devon Branch of Presbyterian Hospital of Philadelphia. Devon, Pa.

1904. Landis, Henry R. M., M.D., Director of the Clinical and Sociological Departments of the Henry Phipps Institute of the University of Pennsylvania; Assistant Professor in Medicine in the University of Pennsylvania; Visiting Physician to the White Haven Sanatorium. 11 S. Twenty-first St.

1907. Langdon, H. Maxwell, M.D., Instructor in Ophthalmology in the University of Pennsylvania; Assistant Surgeon to the Dispensary for Diseases of the Eye in the University Hospital; Assistant Ophthalmologist to the Orthopædic Hospital; Chief of the Dispensary for Diseases of the Eye of the Presbyterian Hospital. 2018 Chestnut St.

1887. Leaman, Henry, M.D. 832 N. Broad St.

1904. Le Boutillier, Theodore, M.D., Clinical Professor of Pediatrics in the Woman's Medical College of Pennsylvania; Pediatrist to the Woman's College Hospital; Physician to the Philadelphia Hospital for Contagious Diseases and the Babies' Hospital of Philadelphia. 9 S. Twenty-first Street.

1893. Le Conte, Robert G., A.B., M.D., Surgeon to the Pennsylvania and the Bryn Mawr Hospitals; Consulting Surgeon to Germantown and Gynecean Hospitals. 1530 Locust Street.

1908. Lee, Walter Estell, M.D., Surgeon to the Gynecological Dispensary of the Pennsylvania Hospital; Assistant Surgeon to the Germantown and Bryn Mawr Hospitals; Surgeon to the Dispensary of the Episcopal Hospital and Children's Hospital; Surgeon to the Glen Mills Schools. 905 Pine St.

ELECTED

1903. Leffmann, Henry, A.M., M.D., D.D.S., Ph.D., Professor of Chemistry in the Woman's Medical College of Pennsylvania; Honorary Professor of Chemistry in the Wagner Free Institute of Science; Pathological Chemist to the Jefferson Medical College Hospital. 1839 N. Seventeenth St.

1892. Leidy, Joseph, M.D., Officer l'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1319 Locust St.

1909. L'Engle, Edward M., M.D. Jacksonville, Fla.

1915. Lewis, Fielding O., M.D., Associate in Laryngology in the Jefferson Medical College Hospital; Operating Clinical Chief of the Laryngological Dispensary and Clinical Assistant of the Otological Dispensary of the Jefferson Medical College Hospital; Laryngologist of the Philadelphia General Hospital. 261 S. Seventeenth St.

1877. Lewis, Morris J., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Pennsylvania Hospital. 1316 Locust St.

1911. Lewis, Paul A., M.D., Director of the Pathological Department of the Henry Phipps Institute of the University of Pennsylvania; Assistant Professor of Pathology in the University of Pennsylvania; Director of the Ayer Clinical Laboratory of the Pennsylvania Hospital. Henry Phipps Institute. Seventh and Lombard Streets.

1904. Lindauer, Eugene, M.D., Instructor of Neurology in the Medico-Chirurgical Hospital; Associate in Clinical Medicine in the Philadelphia Polyclinic; Assistant Neurologist to the Philadelphia General Hospital. 2018 N. Thirty-second St.

1886. Lloyd, J. Hendrie, A.M. (Princeton), M.D., Neurologist to the Philadelphia General Hospital, and to the Methodist Episcopal Hospital; Consulting Neurologist to the State Asylum for the Chronic Insane at Wernersville, and to the Pennsylvania Training School for Feeble-minded Children at Elwyn. 116 S. Twenty-first St.

1907. Lodholz, Edward, M.D., Demonstrator of Physiology in the University of Pennsylvania. 3103 Diamond St.

1893. Longaker, Daniel, M.D., Obstetrician to the Kensington Hospital for Women and Visiting Obstetrician to the Jewish Maternity Hospital. 1402 N. Sixteenth St.

1907. Loux, Hiram R., M.D., Professor of Genito-Urinary Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General Hospital. 1819 Walnut St.

1914. Lyon, B. B. Vincent, A.B. (Williams Coll.), M.D. 1901 Pine St.

ELECTED

1900. McCarthy, Daniel J., M.D., Professor of Medical Jurisprudence (George B. Wood Foundation) in the University of Pennsylvania; Neurologist to the Philadelphia General and St. Agnes' Hospitals, and to the Henry Phipps Institute. 2025 Walnut St.

*1903. McConnell, Guthrie, M.D., Director of the Clinical Laboratory of the Waterloo Medical Society; Deputy State Bacteriologist for Waterloo; Assistant Surgeon, Medical Reserve Corps, U. S. N. 508 South St., Waterloo, Iowa.

1913. McCrae, Thomas, B.A., M.D., F.R.C.P. (Lond.), Professor of Medicine in the Jefferson Medical College; Physician to the Jefferson and Pennsylvania Hospitals. 1627 Spruce Street.

1895. McFarland, Joseph, M.D., Sc.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College; Pathologist to the Medico-Chirurgical Hospital and to the Philadelphia General Hospital. 442 W. Stafford St., Germantown.

1913. McGlinn, John A., B.A., M.D., Assistant Professor of Obstetrics in the Medico-Chirurgical College; Assistant Obstetrician to the Medico-Chirurgical Hospital; Gynecologist to St. Agnes' Hospital. 113 S. Twentieth St.

1905. McKenzie, Robert Tait, A.B., M.D., Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 26 S. Twenty-first St.

1916. McKnight, Howard A., A.B., M.D., Surgeon of Out-patient Department, St. Mary's Hospital; Assistant Surgeon of Out-patient Department, Polyclinic Hospital. 241 S. Thirteenth St.

1915. McLean, John D., M.D. 1538 S. Broad St.

*1900. McReynolds, Robert Phillips, M.D. 213 S. Broadway, Los Angeles, Cal.

1886. MacCoy, Alexander W., M.D. Consulting Laryngologist to the Bryn Mawr Hospital. 216 S. Fifteenth St.

1910. Mackinney, William H., M.D., Assistant Surgeon to the Dispensary for Genito-urinary Diseases, University Hospital; Assistant in the Urological Dispensary of the German Hospital. 1701 Chestnut St.

1914. Maier, F. Hurst, M.D., Associate in Gynecology to the Jefferson Medical College; Gynecologist to St. Joseph's Hospital. 2035 Chestnut St.

ELECTED

1913. Major, C. Percy, M.D., Physician to the Dispensary of the Germantown Hospital; Pediatrist to the Dispensary of the Germantown Hospital; Pediatrist to the Abington Memorial Hospital. Tenth and Oak Lane.

1896. Makuen, G. Hudson, M.D., Professor of Defects of Speech in the Philadelphia Polyclinic; Consultant Laryngologist and Otologist to the Roosevelt and Frederick Douglass Hospitals; Consultant on Defects of Speech to the New Jersey Training School for Feeble-minded Children. 1627 Walnut Street.

1913. Manges, Willis F., M.D., Röntgenologist to the Jefferson Hospital; Director of the Röntgen Ray Laboratory in the Philadelphia General Hospital. 264 S. Sixteenth St.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia General Hospital; Laryngologist and Otologist to St. Joseph's Hospital. 1819 Spruce St.

1893. Marshall, John, M.D., Nat.Sc.D. (Tübingen), LL.D. Professor of Chemistry and Toxicology in the University of Pennsylvania. 1718 Pine St.

1889. Martin, Edward, M.D., John Rhea Barton Professor of Surgery in the University of Pennsylvania; Surgeon to the University, Howard, Philadelphia General, and Bryn Mawr Hospitals. 1506 Locust St.

1885. Mays, Thomas J., M.D. 1829 Spruce St.

*1868. Mears, J. Ewing, A.M., M.D., LL.D. 1535 Land Title Building, Broad and Sansom Sts.

*1911. Meigs, Edward Browning, A.B., M.D., Physiologist in the Dairy Division of the United States Department of Agriculture. 1722 H St., N. W., Washington, D. C.

1914. Mencke, J. Bernhard, A.B., M.D., Assistant Surgeon to the Out-patient Department of the German Hospital; Assistant Surgeon to the Stetson Hospital. 908 N. Sixteenth Street.

1914. Merrill, Wm. Jackson, M.D., 1927 Chestnut St.

*1894. Miller, D. J. Milton, M.D., Associate Physician to the Children's Hospital, Philadelphia; Pediatrist to the Bamberger Home for Invalid Children, Longport, N. J. N. W. Cor. Pacific and California Aves., Atlantic City, N. J.

1910. Miller, Morris Booth, M.D., Professor of Surgery in the Philadelphia Polyclinic; Surgeon to the Douglass Memorial Hospital; Assistant Surgeon to the Philadelphia General Hospital. 2117 Pine St.

ELECTED

1881. Mills, Charles K., M.D., LL.D., Emeritus Professor of Neurology in University of Pennsylvania; Neurologist to Philadelphia General Hospital; Consulting Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1909 Chestnut Street.

1904. Mitchell, Charles F., M.D., Surgeon to the Germantown, Bryn Mawr, and Chestnut Hill Hospitals, and to the Outpatient Department of the Pennsylvania Hospital. 342 S. Fifteenth St.

†1888. Mitchell, John K., M.D., Attending Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Neurologist to the Presbyterian Hospital; Attending Physician to the Pennsylvania Training School for Feebleminded Children. 1730 Spruce St.

1908. Montgomery, Charles M., A.B., M.D., Instructor in the Henry Phipps Institute (University of Pennsylvania); Physician to the Dispensaries of the Pennsylvania and St. Agnes' Hospitals. 2210 Locust St.

1882. Montgomery, Edward E., A.M., M.D., LL.D., Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Jefferson Medical College Hospital and to St. Joseph's Hospital. 1426 Spruce St.

1886. Morris, Caspar, M.D. 2050 Locust St.

1893. Morris, Elliston J., M.D., Physician to the Episcopal Hospital and to the Midnight Mission. 128 S. Eighteenth St.

1883. Morris, Henry, M.D., Professor of Anatomy in the Woman's Medical College of Pennsylvania; Senior Visiting Physician to St. Joseph's Hospital. 313 S. Sixteenth St.

°1856. Morris, J. Cheston, M.D. 1514 Spruce St.

1906. Morrison, William H., M.D. 8021 Frankford Ave.

1897. Morton, Samuel W., M.D. 1933 Chestnut St.

1905. Müller, George P., M.D., Associate in Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Professor of Surgery in the Philadelphia Polyclinic; Surgeon to St. Agnes' Hospital; Consulting Surgeon to the Chester County Hospital. 1729 Pine St.

1915. Musser, John H., Jr., B.S., M.D., Associate in Medicine in the University of Pennsylvania; Physician to the Philadelphia General Hospital; Physician in Charge, Medical Dispensary, University Hospital; Dispensary Chief and Assistant in the Presbyterian Hospital. 121 S. Twentieth Street.

ELECTED

1905. Mutschler, Louis H., M.D., Surgeon to the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital. 2030 Tioga St.

*1896. Myers, T. D., M.D. 321 Story Building, Los Angeles, Cal.

1902. Nassau, Charles F., M.D., LL.D., Assistant Professor of Surgery in the Jefferson Medical College; Surgeon to St. Joseph's Hospital; Consulting Surgeon to the Frankford Hospital; Assistant Surgeon to the Jefferson Medical College Hospital. 1831 Chestnut St.

1886. Neff, Joseph S., A.M., M.D., LL.D., D.P.H. Narbeth, Pa.

1887. Neilson, Thomas Rundle, A.M., M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Professor of Genito-urinary Surgery in the University of Pennsylvania. 1937 Chestnut St.

1905. Newcomet, William S., M.D. 3501 Baring St.

1905. Newlin, Arthur, B.S., M.D., Physician to the Pennsylvania Hospital; Physician to the Dispensary of the Children's Hospital; Assistant Physician to the Orthopædic Hospital. 1804 Pine St.

1899. Nicholson, William Rufus, A.B., M.D., Gynecologist to the Polyclinic and Methodist Episcopal Hospitals; Obstetrician to the Presbyterian Hospital; Associate in Obstetrics in the University of Pennsylvania. 1731 Pine St.

*1889. Noble, Charles P., M.D. Easton, Md.

1898. Nolan, Edward J., M.D., Sc.D., Recording Secretary and Librarian of the Academy of Natural Sciences of Philadelphia. 825 N. Twentieth St.

1905. Norris, Charles C., M.D., Instructor in Gynecology in the University of Pennsylvania; Physician to the Maternity Hospital; Consultant Gynecologist and Obstetrician to the Henry Phipps Institute of the University of Pennsylvania; Assistant Gynecologist to the University Hospital. 1503 Locust St.

1905. Norris, George William, A.B., M.D., Assistant Professor of Medicine in the University of Pennsylvania; Physician to the Pennsylvania Hospital; Assistant Physician to the University Hospital. 1530 Locust St.

*1901. Norris, Henry, M.D., Surgeon to the Rutherford Hospital. Rutherfordton, N. C.

*1865. Norris, Isaac, M.D. Fairhill, Bryn Mawr, Pa.

ELECTED

1892. Norris, Richard C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician in Charge of the Preston Retreat; Visiting Obstetrician to the Philadelphia General Hospital; Gynecologist to the Methodist Episcopal Hospital, and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

1913. O'Neal, Alexander H., A.B., A.M., M.D., Physician to the Cathcart and Richardson Homes, Devon; Anesthetizer to the Bryn Mawr Hospital. St. Davids, Pa.

°1885. Osler, Sir William, Bart., M.D., Regius Professor of Medicine in Oxford University, England. No. 7, Norham Gardens, Oxford, England.

1903. Ostheimer, Maurice, A.B., M.D., Associate in Pediatrics in the University of Pennsylvania; Visiting Physician to the Philadelphia Hospital for Contagious Diseases; Physician-in-Charge of the Children's Dispensary and Assistant Visiting Pediatric Physician, University Hospital; Physician to the Medical Dispensary of the Children's Hospital. 2202 De Lancey St.

1913. Outerbridge, George W., A.B., M.D., Instructor in Gynecology and in Surgical Pathology in the University of Pennsylvania; Assistant Gynecologist to the Methodist Hospital and Assistant Surgeon to the Gynecean Hospital. 2040 Chestnut St.

1915. Owen, Hubley R., M.D., Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the Orthopædic Hospital; Chief Surgeon of the Bureaus of Police and Fire, Philadelphia; Assistant Surgeon of the Medical Reserve Corps of the U. S. Navy. 2046 Pine St.

1897. Packard, Francis R., M.D., Surgeon to the Out-patient Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Laryngologist to the Children's Hospital of Philadelphia; Consulting Aurist to the Bryn Mawr Hospital. 304 S. Nineteenth St.

1898. Page, Henry F., M.D., Assistant Physician to the German Hospital and Physician to the Medical Dispensary of the same; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania. 315 S. Sixteenth St.

1906. Pancoast, Henry K., M.D., Professor of Röntgenology in the University of Pennsylvania and Röntgenologist to the University Hospital. Bala, Pa.

ELECTED

1909. Parish, Benjamin D., B.S., M.D., Assistant Instructor in Otology in the University of Pennsylvania; Assistant Surgeon to the Dispensary for Diseases of the Ear, University Hospital; Aurist and Laryngologist to St. Agnes' Hospital. 29 S. Nineteenth St.

1899. Parke, William E., M.D., Consulting Obstetrician to the Episcopal Hospital; Associate Surgeon to the Kensington Hospital for Women; Gynecologist to the Frankford Hospital. 1739 N. Seventeenth St.

1910. Patterson, Ross Vernet, M.D., Dean, Jefferson Medical College; Assistant Professor of Medicine in the Jefferson Medical College; Physician to the Philadelphia Hospital; Assistant Physician to the Jefferson Hospital. 340 S. Sixteenth St.

1903. Pearce, Richard M., M.D., Sc.D., Professor of Research Medicine in the University of Pennsylvania. 2114 De Lancey Place.

1909. Pemberton, Ralph, M.S., M.D., Visiting Physician to the Presbyterian Hospital; Director of the Department of Clinical Chemistry in the Pathological Laboratory of the Presbyterian Hospital. 2224 Locust St.

†1889. Penrose, Charles Bingham, M.D., Ph.D. (Harvard), Formerly Professor of Gynecology in the University of Pennsylvania. 1720 Spruce St.

1914. Pepper, O. H. Perry, B.S. (Univ. of Penna.), M.D. 1811 Spruce St.

†1902. Pepper, William, M.D., Dean of the School of Medicine, and Assistant Professor of Clinical Pathology in the University of Pennsylvania; Physician to the Philadelphia General Hospital; Assistant Physician to the University Hospital. 1811 Spruce St.

1916. Percival, Milton Fraser, M.D. 2332 S. Broad St.

1912. Petty, Orlando H., B.S., A.M., M.D., Instructor in Medicine in the Jefferson Medical College; Pathologist and Assistant Physician to St. Timothy's Hospital; Physician to the Kensington Dispensary for the Treatment of Tuberculosis. 6215 Ridge Ave.

1905. Pfahler, George E., M.D., Director of the Röntgen Ray Laboratory of Medico-Chirurgical Hospital. 1321 Spruce St.

1915. Pfeiffer, Damon B., A.B., M.D., Instructor in Surgery, University of Pennsylvania; Assistant Surgeon, University Hospital; Pathologist to the German Hospital; Director of the Clinical Laboratory, Presbyterian Hospital. 2028 Pine St.

ELECTED

1907. Pfromm, George W., Ph.G., M.D., Assistant Physician to the American Stomach Hospital; Consulting Physician to the German Protestant Home for the Aged. 1431 N. Fifteenth St.

1907. Phillips, Horace, M.D., Second Assistant Physician to the Pennsylvania Hospital for the Insane; Visiting Physician to the Eastern Penitentiary of Pennsylvania. 4713 Baltimore Ave.

1883. Piersol, George A., M.D., Sc.D., Professor of Anatomy in the University of Pennsylvania. 4724 Chester Ave.

1911. Piersol, George Morris, B.S., M.D., Associate in Medicine in the University of Pennsylvania; Physician to the Episcopal Hospital; Assistant Physician to the University Hospital; Assistant Physician to the Philadelphia General Hospital. 1913 Spruce St.

1905. Pitfield, Robert L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1896. Posey, William Campbell, M.D., Surgeon to the Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmic Surgeon to the Howard Hospital; Ophthalmologist to the Pennsylvania Hospital for Epileptics. 2049 Chestnut St.

1899. Potts, Charles S., M.D., Professor of Neurology in the Medico-Chirurgical College; Neurologist to the Philadelphia General Hospital; Consultant to the Insane Department of the Philadelphia General Hospital; Consultant to the Hospital for the Insane, Atlantic County, New Jersey. 2018 Chestnut St.

1907. Price, George E., M.D., Associate Professor of Nervous and Mental Diseases in the Jefferson Medical College; Neurologist to the Philadelphia General Hospital. 1830 S. Rittenhouse Square.

†1903. Pyle, Walter L., A.M., M.D., Assistant Surgeon to the Wills Eye Hospital. 1931 Chestnut St.

1908. Radcliffe, McCluney, A.M. (Lafayette), M.D., LL.D., Ophthalmic Surgeon to the Presbyterian Hospital; Attending Surgeon to the Wills Eye Hospital. 1906 Chestnut St.

1913. Randall, Alexander, A.M., M.D., Assistant Instructor in Surgery in the University of Pennsylvania; Assistant Surgeon in the Genito-Urinary Dispensary of the University of Pennsylvania. Professional Building, Room 401.

ELECTED

1887. Randall, B. Alexander, M.A., M.D., Professor of Otology in the University of Pennsylvania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb, and to St. Timothy's Hospital. 1717 Locust St.

*1904. Ravenel, Mazyck P., M.D., Professor of Preventive Medicine and Bacteriology and Director of the Public Health Laboratory at the University of Missouri, Columbia, Mo.

1897. Rhein, John H. W., M.D., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic and College for Graduates in Medicine; Neurologist to the Howard Hospital; Physician to the Philadelphia Home for Incurables; Bacteriologist to the Pennsylvania Training School for Feeble-minded Children. 1732 Pine St.

1906. Rhein, Robert D., M.D., Chief Physician to the Clinic of the American Hospital for Diseases of the Stomach; Physician to the Philadelphia Home for Incurables; Examining Physician to the White Haven Sanatorium. 2016 Pine Street.

1891. Rhoads, Edward G., M.D. 159 W. Coulter St., Germantown.

1910. Rhoads, Samuel, M.D., Visiting Physician to the Tuberculosis Department of the Philadelphia General Hospital; Consulting Physician to the Germantown Hospital. 152 Schoolhouse Lane, Germantown.

1898. Riesman, David, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to the Philadelphia General and the Jewish Hospitals. 1715 Spruce St.

1895. Ring, G. Oram, A.M., M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. 2014 Chestnut St.

*1905. Risley, J. Norman, M.D., Assistant Surgeon to the Wills Eye Hospital; Ophthalmologist to the Pennsylvania Training School for Feeble-minded Children. 36 Seventh St., New Bedford, Mass.

1891. Risley, S. D., A.M., M.D., Ph.D., Attending Surgeon to the Wills Eye Hospital; Professor (Emeritus) of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Alumnus Manager of the Univeristy Hospital. 2018 Chestnut St.

ELECTED

†1878. Roberts, John B., M.D., Professor of Surgery in the Philadelphia Polyclinic. 313 S. Seventeenth St.

1899. Roberts, Walter, M.D., Professor of Otology in the Philadelphia Polyclinic; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia General Hospital. 1732 Spruce St.

1903. Robertson, William Egbert, M.D., Professor of Theory and Practice of Medicine and of Clinical Medicine in Temple University; Physician to the Episcopal, Samaritan, and Garretson Hospitals. 327 S. Seventeenth St.

1902. Robinson, James Weir, M.D., Assistant Surgeon to the Presbyterian Hospital. 326 S. Sixteenth St.

1903. Robinson, William Duffield, Ph.G., M.D. 2012 Mount Vernon St.

1912. Rodman, John Stewart, M.D., Lecturer on Surgery in the Medico-Chirurgical College; Assistant Surgeon to the Medico-Chirurgical Hospital; Surgeon to the Dispensary of the Presbyterian Hospital; Assistant Surgeon to the Out-patient Department of the Pennsylvania Hospital. 2106 Walnut St.

1909. Rosenberger, Randle C., M.D., Professor of Hygiene and Bacteriology in the Jefferson Medical College; Professor of Hygiene and Preventive Medicine in the Woman's Medical College of Pennsylvania; Director of the Clinical Laboratory of the Philadelphia General Hospital. 2330 N. Thirteenth St.

1898. Ross, George G., M.D., Assistant Surgeon to the German Hospital and Surgeon to the Out-patient Department of the same; Surgeon to the Germantown Hospital; Surgeon to the Stetson Hospital; Instructor in Surgery in the University of Pennsylvania. 1721 Spruce St.

*1907. Royer, B. Franklin, M.D. Donaldson Bldg., Harrisburg, Pa.

†1905. Rugh, James Torrance, A.B., M.D., Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Orthopedic Surgeon to the Jefferson Medical College; Orthopedic Surgeon to the Methodist and the Philadelphia General Hospitals. 1616 Spruce St.

1897. Sailer, Joseph, Ph.B., M.D., Professor of Clinical Medicine in Universtiy of Pennsylvania; Physician to the Philadelphia General, the University, and Presbyterian Hospitals. 1830 Spruce St.

ELECTED

1900. Sajous, Charles E. de M., M.D., B.Lett., LL.D., Knight of the Legion of Honor, and Officer of the Academy of France; Corresponding Member of the Society of Public Medicine of Belgium. 2043 Walnut St.

1905. Sartain, Paul J., M.D. 212 W. Logan Square.

1908. Sautter, Albert C., M.D., Assistant in the Dispensary for Diseases of the Eye in the University Hospital; Assistant in the Eye Dispensary of the German Hospital. 1421 Locust St.

1906. Saylor, Edwin S., M.D., Chief Ophthalmic Surgeon to the Charity Hospital of Philadelphia, and to the Department Eye and Ear, American Hospital for Diseases of the Stomach. 2005 Chestnut St.

*1910. Scarlet, Rufus B., M.D., Laryngologist to the Home for Consumptives at Chestnut Hill; Assistant in the Department for Diseases of the Ear, Throat, and Nose of the Pennsylvania Hospital; Instructor in Diseases of the Nose and Throat in the Philadelphia Polyclinic and College for Graduates in Medicine. 78 N. Clinton Ave., Trenton, N. J.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Infectious Eruptive Diseases in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Physician to the Municipal Hospital for Infectious Diseases. 1922 Spruce St.

1887. de Schweinitz, George Edmund, A.M., M.D., LL.D., Professor of Ophthalmology in the University of Pennsylvania; Consulting Ophthalmic Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; The Philadelphia Polyclinic and School for Graduates in Medicine, and the Philadelphia General Hospital; Ophthalmic Surgeon to the University Hospital. 1705 Walnut St.

*1913. de Schweinitz, George Lord, B.S., M.D. 169 East Broad St., Bethlehem, Pa.

1910. Schwenk, Peter N. K., M.A., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital; Attending Surgeon to the Wills Eye Hospital. 1417 N. Broad Street.

1892. Seiss, Ralph W., M.D., Professor of Otology in the Philadelphia Polyclinic; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb. 255 S. Seventeenth St.

ELECTED

1908. Shannon, Charles E. G., A.B., M.D., Instructor in Ophthalmology in the Jefferson Medical College; Assistant in the Ophthalmological Clinic at the Jefferson Medical College Hospital; Ophthalmologist to the Seybert Institution. 1633 Spruce St.

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1906. Shields, William G., M.D., Dermatologist to Germantown Hospital; Chief of Dermatological Clinic and Assistant Physician to the Jewish Hospital. 414 School Lane, Germantown.

1890. Shoemaker, George Erety, A.M., M.D., Gynecologist to the Presbyterian Hospital and to the Pennsylvania Epileptic Hospital and Colony Farm. 1831 Chestnut St.

*1908. Shoemaker, Harlan, A.B., M.D., Lecturer in Surgery, University of Southern California, Surgeon to Los Angeles County Hospital, Surgeon to Washington Street Clinic. 621 Marsh Strong Building, Los Angeles, Cal.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms; Consulting Physician to the Southern Home for Destitute Children; Assistant Physician to the German Hospital; Physician to the Out-patient Department of the German Hospital. 2011 Chestnut St.

†1896. Shoemaker, William T., M.D., Associate Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania; Ophthalmic Surgeon to the Germantown and German Hospitals; Attending Surgeon to the Eye Department of the Pennsylvania Hospital; Consulting Ophthalmologist to the Pennsylvania Institution for the Deaf and Dumb, and to the Southern Home for Destitute Children. 109 S. Twentieth St.

1900. Shumway, Edward Adams, B.S., M.D., Ophthalmic Surgeon to the Philadelphia General Hospital; Ophthalmic Surgeon to the Children's Hospital; Instructor in Ophthalmology in the University of Pennsylvania, and Assistant Ophthalmic Surgeon to the University Hospital; Ophthalmic Surgeon to the German Hospital. 2007 Chestnut St.

1903. Sinclair, John Falconer, M.D., Physician to the Medical Dispensary of the Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

ELECTED

1907. Sinkler, Francis Wharton, A.B., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to the Episcopal Hospital. 1606 Walnut St.

1902. Siter, E. Hollingsworth, M.D., Instructor in Genito-urinary Diseases in the University of Pennsylvania; Genito-urinary Surgeon to the Philadelphia General Hospital; Surgeon-in-Charge of the Genito-urinary Dispensary of the University Hospital; Consulting Genito-Urinary Surgeon to the Eastern Penitentiary, and to the Philadelphia County Prison. 1818 S. Rittenhouse Square.

1904. Skillern, Penn-Gaskell, M.D. 241 S. Thirteenth St.

1904. Smith, Allen J., A.M., M.D., Sc.D. (Penna. Coll.), LL.D. (McGill Univ.), Professor of Pathology and of Comparative Pathology, and Director of Courses in Tropical Medicine in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1905. Smith, S. MacCuen, M.D., Professor of Otology in the Jefferson Medical College; Aurist and Laryngologist to the Germantown Hospital; Aurist to the Jewish Hospital; Consulting Aurist to the Oncologic Hospital. 1429 Spruce Street.

1908. Speese, John, M.D., Instructor in Surgery and Surgical Pathology in the University of Pennsylvania; Associate in Surgery in the Philadelphia Polyclinic; Surgeon to the Children's Hospital; Assistant Surgeon to the Presbyterian and Polyclinic Hospitals. 2206 Locust St.

1895. Spellissy, Joseph M., A.M., M.D., Visiting Surgeon to St. Joseph's and to the Methodist Episcopal Hospitals; Assistant Surgeon to the Orthopedic Department of the University Hospital. 317 S. Fifteenth St.

1897. Spiller, William G., M.D., Professor of Neurology in the University of Pennsylvania; Clinical Professor of Nervous Diseases in the Woman's Medical College of Pennsylvania; Neurologist to the Philadelphia General Hospital; Consultant Neurologist to the Pennsylvania Hospital. 4409 Pine St.

1894. Stahl, B. Franklin, B.S., Ph.G., M.D., Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Associate in Medicine and Lecturer on Dietetics of the Sick in the University of Pennsylvania; Visiting Physician to St. Agnes' and the Philadelphia General Hospitals. 1727 Pine St.

1909. Starbuck, J. Clinton, M.D. 42 E. Washington St., Media, Pa.

ELECTED

*1875. Starr, Louis, M.D., LL.D. (Haverford). The Vanderbilt Hotel, 34th St. and Park Ave., New York City.

1912. Stauffer, Nathan Pennypacker, D.D.S., M.D., Laryngologist and Otologist to the Dispensary of the Presbyterian Hospital; to the Pennsylvania Hospital; to the Philadelphia General Hospital, Tubercular Department; to the Home for Crippled Children. 218 S. Twentieth St.

1910. Stellwagen, Thomas C., Jr., M.D., Chief Clinical Assistant in the Out-patient Surgical Department of the Jefferson Medical College Hospital. 200 Professional Bldg., 1821 Chestnut St.

1884. Stelwagon, Henry W., M.D., Ph.D., Professor of Dermatology in the Jefferson Medical College. 1634 Spruce St.

1895. Stengel, Alfred, M.D., Sc.D., Professor of Medicine in the University of Pennsylvania; Physician to the University Hospital and the Pennsylvania Hospital. 1728 Spruce Street.

1901. Stevens, Arthur A., M.D., Professor of Materia Medica, Therapeutics, and Clinical Medicine in the Woman's Medical College of Pennsylvania; Lecturer on Physical Diagnosis in the University of Pennsylvania; Physician to the Episcopal and St. Agnes' Hospitals. 314 S. Sixteenth St.

1902. Stewart, Francis T., M.D., Professor of Clinical Surgery in the Jefferson Medical College; Surgeon to the Germantown Hospital; Surgeon to the Pennsylvania Hospital. 311 S. Twelfth St.

1914. Stewart, Thomas S., B.S. (Univ. of Penna.), M.D. 301 S. Eighteenth St.

1898. Stout, George C., M.D., Professor of Otology in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society, and the William Penn Charter School. 1611 Walnut St.

1884. Stryker, Samuel S., M.D., Physician to the Presbyterian Hospital. 3833 Walnut St.

*1900. Swan, John M., M.D. 457 Park Avenue, Rochester, N. Y.

1898. Sweet, William M., M.D., Clinical Professor of Ophthalmology in the Jefferson Medical College, and Ophthalmic Surgeon to the Jefferson Medical College Hospital; Professor of Diseases of the Eye in the Philadelphia Polyclinic; Attending Surgeon to the Wills Eye Hospital. 1205 Spruce St.

ELECTED

1900. Talley, James Ely, A.B., M.D., Visiting Physician to the Presbyterian and Methodist Episcopal Hospitals; Consulting Physician to the Eastern Penitentiary. 218 S. Twentieth Street.

1911. Taylor, Alonzo Englebert, M.D., Rush Professor of Physiological Chemistry in the University of Pennsylvania. 4522 Locust St.

1886. Taylor, John Madison, A.B. and A.M. (Princeton), M.D., Professor of Applied Therapeutics in the Temple University; Editor *Monthly Cyclopedia of Practical Medicine;* Consulting Physician to the Elwyn, Pa., and the Vineland, N. J., Training Schools for Feeble-minded Children. 1504 Pine St.

1887. Taylor, William J., M.D., Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes' Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women. 1825 Pine St.

1886. Taylor, William L., M.D. 1340 N. Twelfth St.

†1910. Thomas, Benjamin A., A.M., M.D., Professor of Genito-urinary Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Instructor in Surgery in the University of Pennsylvania; Surgeon-in-Chief to the Outpatient Department of the University Hospital. 116 S. Nineteenth St.

1867. Thomas, Charles Hermon, M.D. 3634 Chestnut St.

1912. Thomas, Frank Wister, A.M., M.D., Visiting Physician to "Buttercup Cottage;" Consulting Physician to the Germantown Hospital. 27 E. Mt. Airy Ave.

1907. Thomas, Thomas Turner, M.D., Associate Professor of Applied Anatomy, and Associate in Surgery in the University of Pennsylvania; Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the University Hospital. 2005 Chestnut St.

1897. Thomson, A. G., M.D., Ophthalmic Surgeon to the Pennsylvania Railroad Company. 724 Stock Exchange Bldg.

1896. Thorington, James, A.M., M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmic Surgeon to the Presbyterian Hospital; Ophthalmologist to the Ellwyn, Pa., Training School for Feeble-minded Children. 2031 Chestnut St.

1898. Thornton, Edward Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 1331 Pine St.

ELECTED

1912. Torrey, Robert G., M.D., Assistant Physician to the Philadelphia General Hospital; Physician to the State Tuberculosis Dispensary. 1716 Locust St.

1896. Toulmin, Harry, M.D., Medical Director of the Penn Mutual Life Insurance Company. 925 Chestnut St.

1908. Tracy, Stephen E., M.D., Gynecologist to the Stetson Hospital. 1527 Spruce St.

1901. Tucker, Henry, M.D., Genito-urinary Surgeon to the Philadelphia General Hospital; Curator of the Academy of Natural Sciences of Philadelphia. 2000 Pine St.

†1894. Tunis, Joseph Price, M.D., Clinical Assistant to the Nose and Throat Dispensary of the Polyclinic Hospital. San Ysidro Ranch, Santa Barbara, California.

1901. Turner, John B., M.D. 1833 Chestnut St.

1866. Tyson, James, M.D., LL.D., Emeritus Professor of Medicine in the University of Pennsylvania and late Physician to the Hospital of the University of Pennsylvania, and to the Pennsylvania Hospital. 1506 Spruce St.

1897. Tyson, T. Mellor, M.D., Physician to the Philadelphia General Hospital; Physician to the Rush Hospital, the Philadelphia Lying-in-Charity Hospital, and the Children's Aid Society of Philadelphia. 1506 Spruce St.

1907. Ullom, Josephus Tucker, M.A., M.D., Member of the Staff of the Henry Phipps Institute, Visiting Physician to the Chestnut Hill Hospital. 24 Carpenter St., Germantown.

1913. Vail, William Penn., B.S., M.S., M.D., Laryngologist to the Pennsylvania Institution for the Instruction of the Blind; Laryngologist to the Department for Tuberculosis of the Philadelphia General Hospital; Assistant Laryngologist and Otologist to the Out-patient Department of the Pennsylvania Hospital; Assistant Laryngologist to the Children's Hospital. 1700 Walnut St.

°1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Children's Hospital. 1831 Chestnut St.

1903. Van Pelt, William Turner, M.D. Consulting Ophthalmologist to the Episcopal Hospital. 1100 Widener Building.

ELECTED

1893. Vansant, Eugene Larue, M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Visiting Physician to the Throat, Nose, and Ear Department of the Howard Hospital. 1929 Chestnut St.

1912. Vaux, Norris Wistar, M.D., Surgeon to the Chestnut Hill Hospital; Surgeon to the Dispensary of the Germantown Hospital. 8901 Germantown Ave., Chestnut Hill.

*1897. Veasey, Clarence A., A.M., M.D. Suite 404, Paulsen Building, Spokane, Wash.

†1883. Vinton, Charles Harrod, A.M., M.D. P. O. Box 464, Atlantic City, N. J.

1903. Wadsworth, William Scott, M.D. 3914 Baltimore Ave.

1906. Walker, John K., M.D., Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Out-patient Department of the Pennsylvania Hospital. 1915 Rittenhouse St.

1907. Walker, Warren, M.D., Dermatologist to the Episcopal and Howard Hospitals; Assistant Dermatologist to the Pennsylvania Hospital. 246 S. Twenty-second St.

1904. Walsh, Joseph, A.M., M.D., Visiting Physician to and Medical Director of the White Haven Sanatorium; Medical Director of St. Agnes' Hospital. 2026 Chestnut St.

1910. Ward, E. Tillson, A.M., M.D. 1415 S. Broad St.

*1893. Warren, Joseph W., M.D., Department of Health. Harrisburg, Pa.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to Home for Incurables. 126 S. Eighteenth Street.

1886. Watson, Edward W., M.D., Physician to the Magdalene Home. 38 S. Nineteenth St.

1903. Weber, Charles H., M.D., Physician to the Dispensary of the Children's Hospital. 2048 Pine St.

1906. Weisenburg, Theodore H., M.D., Professor of Clinical Neurology and of Neuropathology in the Medico-Chirurgical College; Neurologist to the Philadelphia General Hospital; Consulting Neurologist to the State Hospital for the Insane at Norristown, and to the State Hospital for the Feeble-minded and Epileptic at Spring City. 1909 Chestnut St.

ELECTED

1883. Welch, William M., M.D., Chief Diagnostician to the Bureau of Health, and Consulting Physician to the Municipal Hospital for Contagious Diseases; Consulting Physician to the Northern Dispensary and the Northern Home for Friendless Children. 1411 Jefferson St.

1916. Welker, Henry C., Ph.B., A.M., M.D., Surgeon to Charity Hospital, Norristown, Pa.; Consulting Surgeon to the Norristown State Hospital for the Insane. 831 De Kalb St., Norristown, Pa.

1913. Wells, P. Frailey, A.M., M.D., Ph.D. S. W. Cor. Fortieth and Brown Sts.

1897. Wells, William H., M.D., Assistant Professor of Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson Medical College Hospital; Pediatrician to the Willing Day Nursery; Pediatrician to the Charity Hospital. 1135 Spruce St.

1893. Westcott, Thompson S., M.D., Associate in Diseases of Children in the University of Pennsylvania; Pediatrist to the Methodist Episcopal Hospital; Assistant Physician to the Children's Hospital; Pediatrist to the Jewish Hospital. 1720 Pine St.

1884. Wharton, Henry R., M.D., Surgeon to the Presbyterian and the Children's Hospitals; Surgeon to Girard College; Consulting Surgeon to the Bryn Mawr Hospital, the Chestnut Hill Hospital, St. Christopher's Hospital for Children, the Pennsylvania Institution for the Deaf and Dumb, and the Pennsylvania Institution for the Blind. 1725 Spruce Street.

1901. White, Courtland Y., M.D., Director of the Pathological Laboratories of the Episcopal Hospital; Pathologist to the Children's Hospital and to the Kensington Hospital for Women; Chief Bacteriologist to the Bureau of Health, Department of Health and Charities, Philadelphia. 1808 Diamond St.

1905. Whiteway, Harold M., M.D. 1924 Chestnut St.

1898. Whiting, Albert D., M.D., Surgeon to the Germantown Hospital; Assistant Surgeon to the German Hospital; Assistant Surgeon to the University Hospital; Instructor in Surgery in the University of Pennsylvania. 1523 Spruce Street.

1914. Willard, de Forest P., B.S. (Univ. of Penna.), M.D. 1933 Chestnut St.

ELECTED

1907. Williams, Carl, B.S., M.D., Ophthalmic Surgeon to the Germantown Hospital; Instructor in Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Pennsylvania Institution for the Deaf and Dumb. School Lane and Greene Sts., Germantown.

1915. Willits, Charles H., A.M., M.D., Medical Director of the Provident Life and Trust Company. Rittenhouse Hotel, Twenty-second and Chestnut Sts.

1916. Wilmer, Harry B., M.D., Assistant Instructor in Medicine, University of Pennsylvania; Assistant Visiting Physician and Assistant Neurologist to the Germantown Hospital; Visiting Chief to the Dispensary of the Germantown Hospital. 138 W. Walnut Lane, Germantown.

1881. Wilson, H. Augustus, M.D., Professor of Orthopedic Surgery in the Jefferson Medical College; Emeritus Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Consulting Orthopedic Surgeon to the Philadelphia Lying-in Charity Hospital and the Kensington Hospital for Women; Orthopedic Surgeon to St. Agnes' Hospital. 1611 Spruce St.

1874. Wilson, James Cornelius, A.M. (Princeton), M.D., Emeritus Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College; Physician-in-Chief to the German Hospital; Emeritus Physician to the Pennsylvania Hospital; Consulting Physician to the Bryn Mawr Hospital. 1509 Walnut St.

1902. Wilson, Samuel M., M.D. 1517 Arch St.

1897. Wilson, W. Reynolds, M.D. 1709 Spruce St.

1912. Winsor, Henry, M.D., Surgeon to the Dispensary of the Episcopal Hospital; Prosector to the Associate Professor of Applied Anatomy, and Assistant Instructor in Surgery in the University of Pennsylvania. 2046 Pine St.

1904. Wister, James W., M.D., Physician to the Out-patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. Witmer, A. Ferree, M.D. Freeport, Long Island, N. Y.

1893. Wood, Alfred C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General, St. Timothy's, and the Howard Hospitals. 2035 Walnut St.

1900. Wood, George B., M.D., Instructor in Laryngology in the University of Pennsylvania; Assistant in the Clinic for Diseases of the Nose and Throat at the Polyclinic Hospital. 129 S. Eighteenth St.

ELECTED

1865. Wood, Horatio C, M.D., LL.D (Yale, Lafayette and University of Pennsylvania), M. N. A. S., Emeritus Professor of Materia Medica and Therapeutics in the University of Pennsylvania. 4107 Chester Ave.

1903. Wood, Horatio C., Jr., M.D., Professor of Pharmacology and Therapeutics in the Medico-Chirurgical College. 1905 Chestnut St.

1880. Woodbury, Frank, M.D., Secretary to the Committee on Lunacy of the Board of Charities of Pennsylvania. 218 S. Sixteenth St.

*1911. Woods, Andrew H., A.B., M.D., Neurologist to the Canton Hospital and to the Canton Hospital for Women; Acting President and Medical Superintendent of the Canton Christian College. Canton Christian College, Canton, China.

†1897. Woodward, George, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1913. Woodward, W. Wellington, M.D. 26 S. Church St., West Chester, Pa.

1903. Worden, Charles B., M.D., Associate in Diseases of the Stomach and Intestines in the Philadelphia Polyclinic; Physician to the Dispensary of the Presbyterian Hospital; Anesthetizer to the Orthopedic Department of the University Hospital; Physician to the Presbyterian Orphanage. 322 S. Sixteenth St.

1889. Young, James K., M.D., Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Associate Professor of Orthopedic Surgery in the University of Pennsylvania. 222 S. Sixteenth St.

1894. Zentmayer, William, M.D., Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Attending Surgeon to the Wills Eye Hospital; Ophthalmologist to the Glen Mills School. 1819 Spruce St.

1899. Ziegler, S. Lewis, A.M., M.D., LL.D., Attending Surgeon to the Wills Eye Hospital; Chief Ophthalmic Surgeon to St. Joseph's Hospital; Membre Société Française d'Ophthalmologie; Director of Public Health and Charities of Philadelphia. 1625 Walnut St.

1887. Ziegler, Walter M. L., A.M., M D. 1418 N. Seventeenth St.

1895. Zimmerman, Mason W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1522 Locust St.

ASSOCIATE FELLOWS

(Limited to Fifty, of whom Twenty may be Foreigners)

American

ELECTED

1911. Abbe, Robert, M.D. 13 W. Fiftieth St., New York City, N. Y.

1909. Billings, Frank, M.D., 122 S. Michigan Boulevard, Chicago, Illinois.

1886. Cheever, David W., M.D., 557 Boylston Street, Boston, Massachusetts.

1893. Councilman, William T., M.D., Harvard Medical College, Boston, Massachusetts.

1909. Crile, George W., M.D., 1021 Prospect Avenue, S. E., Cleveland, Ohio.

1909. Dana, Charles Loomis, M.D., 53 West Fifty-third Street, New York City, New York.

1892. Emmet, Thomas Addis, M.D., 91 Madison Avenue, New York City, New York.

1903. Gorgas, William C., M.D., U. S. A., Washington, D. C.

1891. Jacobi, A., M.D., 19 East Forty-seventh Street, New York City, New York.

1912. McCaw, Lieut. Col. Walter D., M.D. Care of Surgeon-General's Office, Washington, D. C.

1909. Mall, Franklin P., M.D., 1514 Bolton Street, Baltimore, Maryland.

1906. Mayo, William J., M.D., Rochester, Minnesota.

1906. Pilcher, Lewis Stephen, M.D., 145 Gates Avenue, Brooklyn, New York.

1886. Reeve, John C., M.D., LL.D., S. W. corner Third and Wilkinson Streets, Dayton, Ohio.

1906. Shattuck, Frederick C., M.D., 135 Marlborough Street, Boston, Massachusetts.

ELECTED

1894. Warren, J. Collins, M.D., 58 Beacon Street, Boston, Massachusetts.
1894. Weir, Robert F., M.D., 11 East Fifty-fourth Street, New York City, New York.
1892. Welch, William H., M.D., Johns Hopkins Hospital, Baltimore, Maryland.

Foreign

1890. Baccelli, Guido, Rome, Italy.
1908. Bannerman, W. B., M.D., General, I. M. S., 11 Strathearn Place, Edinburgh, Scotland.
1899. Fraser, Sir Thomas R., M.D., LL.D., F.R.C.P., F.R.S., 13 Drumsheigh-Gardens, Edinburgh, Scotland.
1893. von Jaksch, Rudolf, M.D., Prague, Bohemia.
1903. Kocher, Prof. Theodor, M.D., Laupenstrasse 25, Berne, Switzerland.
1912. Lazarus, Paul, M.D., Hindersinstr. 2, Berlin, N. W., Germany.
1909. Macallum, Archibald B., M.A., M.B., Ph.D., Sc.D., LL.D., F.R.S., 59 St. George Street, Toronto, Canada.
1906. Myles, Sir Thomas, M.D., 33 Merion Square, W., Dublin, Ireland.
1898. Roddick, Thomas G., M.D., 80 Union Avenue, Montreal, Canada.
1908. Ross, Major Ronald, M.D., R.A., Liverpool School of Tropical Medicine, Liverpool, England.
1904. Waldeyer, Prof. Wilhelm, M.D., W. 62 Sutherstr. 35, Berlin, Germany.

CORRESPONDING MEMBERS

1880. Carrow, Flemming, M.D., Washington Arcade, Detroit, Michigan.
1885. Rendu, Jean, M.D., Lyons, France.
1915. McIntire, Charles, M.D., Easton, Pennsylvania.
1916. Estes, William Lawrence, M.D., South Bethlehem, Pa.
1916. Guitéras, John, M.D., Havana, Cuba.

NECROLOGICAL LIST

FELLOWS

ROBERT N. WILLSON,	January	1, 1916
WILLIAM L. RODMAN,	March	8, 1916
EMLEN PHYSICK,	March	21, 1916
CHARLES CLAXTON,	April	4, 1916
NORTON DOWNS,	April	15, 1916
J. WILLIAM WHITE,	April	24, 1916
EDWARD L. DUER,	September	6, 1916
ALEXANDER WILLIAMS BIDDLE,	September	19, 1916
ALEXANDER A. UHLE,	October	21, 1916
D. BRADEN KYLE,	October	23, 1916

ASSOCIATE FELLOWS

Sir VICTOR ALEXANDER HADEN HORSLEY, F.R.S.,	July	16, 1916
Sir THOMAS LAUDER BRUNTON, M.D.,	September	16, 1916
L. McLANE TIFFANY, M.D.,	October	23, 1916

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MEMOIR OF DR. MORRIS LONGSTRETH[1]

By JAMES TYSON, M.D.

Dr. Morris Longstreth was born in Philadelphia, February 24, 1846, and died in Barcelona, Spain, September 18, 1914, aged sixty-eight years.

He was the son of Thomas B. and Lydia Noble Longstreth. Thomas B. Longstreth was descended from Bartholomew Longstreth, who came to Pennsylvania from Yorkshire, England, in 1699, and settled near Hatboro, Bucks County, on a farm of one thousand acres. Lydia Noble, his wife, was descended from Abel Noble, who came from Bristol, England, in 1684, and also settled in Bucks County, Pa.

Dr. Longstreth was educated at Friends' Schools and at Haverford College, Pennsylvania, whence he was graduated A.B. in 1864. He also received the same degree from Harvard College in 1866. He studied medicine at the University of Pennsylvania and was graduated M.D. in 1869. He was appointed resident physician at the Pennsylvania Hospital shortly afterward and served the usual term of eighteen months. While resident there he was appointed curator of the Pathological Museum, and during his term he arranged and catalogued the specimens. In 1875 he gave a course of lectures on Pathological Anatomy, illustrated by specimens from the museum. In 1872 he was elected physician to the out-patient department, and in 1879 a member of the visiting medical staff. Shortly afterward, in 1879, he was appointed Professor of Pathological Anatomy in Jefferson Medical

[1] Read March 1, 1916.

College. In 1884 he gave by invitation a course of lectures at Lowell Institute in Boston, selecting for his subject, "Against the Germ Theory of Disease." This was soon after the discovery of the tubercle bacillus by Koch in 1882, and at a time when Koch's views were highly popular, so that Dr. Longstreth's were disappointing to the profession. A perusal of at least one of Dr. Longstreth's more recent papers goes to show, however, that he did not ignore pathogenic bacteria, and especially the tubercle bacillus.[1]

While resident at the Hospital he had given special attention to rheumatic diseases, and his studies led to the request of a medical publisher that he publish them. The outcome was a volume entitled *Rheumatism, Gout, and Some Allied Disorders* (New York: Wm. Wood & Co., 1882; pp. 280). In this work he collected much information and presented very clearly and fairly the various conceptions of the nature of the disease as held at that day. Another paper by Dr. Longstreth which I recall was in association with the late Prof. J. M. Da Costa, entitled "Researches on the State of the Ganglionic Centers in Bright's Disease" (*Am. Jour. Med. Sc.*, N. S., 1880, lxxx, 25).

He resigned his positions at the Pennsylvania Hospital and the Jefferson Medical College in 1895, after which he continued private practice which he began in Philadelphia at the expiration of his term of resident physician at the Pennsylvania Hospital. For a number of years he spent his summers at Bar Harbor, Maine, where he acquired a large and lucrative practice, being consulted by patients from all parts of the United States, attracted by novelties in treatment suggested by his anatomical studies.

One of these anatomical illustrations is seen in the effect of slight obstruction of the circulation in a vein, resulting in distal distention, gradually lessening until finally the vein has disappeared. He instances examples in the hand from wearing tight wristlets, in the legs from elastic garters, and in the feet by tight boots,

[1] See the manuscript paper alluded to at the end of this memoir, which might be entitled "Deleterious Results from Defective Body Position." Dr. Longstreth apparently endorsed it "Circulation." I am informed by a relative of Dr. Longstreth that it was written by Dr. Longstreth some time in 1912.

producing perverted sensation—varying in grade from the slightest to the most severe—changes in the growth of the hair, of the color of the skin in spots, and deterioration in the growth of the nails. The special type of this obstruction he claimed to be the column of slowly moving or oscillating blood in a partially obstructed vein, to which also gravity contributes.

He instances other partial obstructive forces at play acting on the deeper organs of the body, and especially in the pelvic organs of women. Some of these are abnormal position or posture. This results in localized areas of hyperemia, which, causing obstruction in varying degrees, produces also disturbed function. From such a defective body posture favored by gravity and other varieties of obstruction results a multitude of evils and deformities for which Dr. Longstreth sought relief by various mechanical appliances. He advocated the examination of large numbers of persons, their conditions, and life history, and compared these with their effectiveness in living and conducting the affairs of life. Dr. Longstreth was convinced, for example, that a large part of infantile troubles go along with faulty body posture. Similarly does he ascribe the deficiencies of "retarded and delayed scholars," the victims of tuberculosis and other diseases. These are interesting and important subjects, and the modern practice now growing rapidly of examining physically men and women seeking employment is an application of what Dr. Longstreth advocated many years ago. Dr. Longstreth was one of the original members of the Association of American Physicians (1886), a Fellow of the College of Physicians of Philadelphia from 1877, and other scientific societies.

By his death Harvard University and the College of Physicians of Philadelphia were made co-heirs of his residuary estate, the principal in the case of the College to be held and invested and the income thereof to be used in defraying the salaries of the librarian and the assistants in the library, and to the binding of books so far as the income to be derived from said fund may suffice; or should any excess of income accrue beyond that, it may be appropriated and applied as directed by the College for other purposes connected with the library. He desired that

before making the distribution between Harvard University and the College of Physicians all his medical and scientific books, pamphlets, and instruments should be given to the College of Physicians. Approximately, the College will receive between $25,000 and $30,000 from Dr. Longstreth's estate.

The breaking out of the War, in August, 1914, found Dr. and Mrs. Longstreth in Genoa with their friend and relative Mr. Lucian Sharpe. Escape from the confusion of numbers and from possible danger, in a place so near a possible seat of war, seemed easy by way of Barcelona, Spain; but arrival at the latter place about the middle of August found the conditions as to crowding about the same as in Genoa. Mrs. Longstreth had been ailing more or less all the winter previous, and within three or four days after their arrival in Barcelona she died.

Whether the local conditions had anything to do with hurrying Mrs. Longstreth's end we do not know, but Dr. Longstreth's death followed hers within a month. Their affection and interdependence were matters of admiration among their friends, and whatever the chief cause of death in Dr. Longstreth's case, it is reasonable to believe it was materially accelerated by the heartbreaking anxiety and fatigue incident to the care and nursing he gave her. The bodies were brought home for burial.

For most of the information obtained in the sketch of Dr. Longstreth, I am indebted to the executrix of his estate, Miss Agnes Longstreth Taylor, and to the unpublished paper above referred to, also obtained through the kindness of Miss Taylor.

GEORGE MILLER STERNBERG, M.D., LL.D.[1]

Surgeon-General, U. S. Army, 1893 to 1902

A NOTE IN MEMORIAM

By A. C. ABBOTT, M.D.

I am highly honored by your invitation to present a minute on the death of our late fellow, George Miller Sternberg, Surgeon General of the United States Army, retired. In the death of General Sternberg we note the passing of one of America's most active pioneers in modern preventive medicine. He was certainly the first American to be conspicuously identified with the science of bacteriology and to recognize its bearings upon the problems of epidemiology and prevention. His most important investigations were made at a time long antedating the advent of the methods of Koch now invariably employed in bacteriological research; methods that are today the common property of every medical student graduated from our reputable schools. It was my happy privilege to have been associated with Dr. Sternberg in the fall and winter of 1884–85—when his rank was that of major. He was then holding, by courtesy, a Fellowship in the Johns Hopkins University. It was through his influence and kindly interest that my attention was drawn to the problems of public hygiene, and I am grateful for this opportunity to record my deep indebtedness to him. Only those intimately associated with Dr. Sternberg can appreciate his singular individuality—a tireless worker, unsatisfied with anything less than the best;

[1] Read April 5, 1916.

convinced only by arguments emanating from experimental data and always with an eye to the application of results to the welfare of mankind. Busy at all times, but never hurried, his affairs were in such order that confusion was unknown; modest, kindly, generous and patient, it was a privilege and delight to receive the benefit of his counsel.

General Sternberg was born at Hartwick Seminary, Otsego Co., N. Y., on June 8, 1838. He died at his residence in Washington, D. C., on November 3, 1915, at the age of seventy-seven years. He was a descendant of one of the German families from the Palatinate that early settled New York State. He spent his boyhood days and received his preliminary education at Hartwick Seminary where, for a time, his father was the principal, and his grandfather, on the maternal side, was a professor. At the age of sixteen he was a self-supporting school teacher. He began the study of medicine at the age of nineteen under the preceptorship of Dr. Horace Lathrop of Cooperstown, N. Y., and was graduated as Doctor of Medicine from the College of Physicians and Surgeons with the class of 1860. He practised medicine for a time at Elizabeth, N. J.

With the outbreak of the Civil War he decided to enter the army, and in 1861, after due examination, was appointed Assistant Surgeon of the United States Army. He was in active service throughout the war. He was present at the first battle of Bull Run, and at the engagements at Gaine's Mills, Turkey Bridge, and Malvern Hill. He received brevet commission for meritorious services during the war and later the brevet commission of Lieutenant-Colonel for "gallant service in the performance of his professional duties under fire in action against the Indians at Clearwater, Idaho, in 1877."

In an exhaustive notice of General Sternberg's scientific and military career, his friend, Dr. Kober, of Washington, remarks: "Dr. Sternberg has seen more active service on the battlefield and in Indian campaigns than any other medical officer with whose records we are familiar."

But it is not alone on the battlefield that Dr. Sternberg exhib-

ited his fidelity to duty. He was medical officer at Fort Harken, Kansas, in 1867, during an epidemic of Asiatic cholera, and was post surgeon during an outbreak of yellow fever among the troops at Fort Columbus, N. Y., in 1871. It is probable that impressions made by the ravages of those diseases had much to do with deciding Dr. Sternberg in the course that his subsequent medical activities were to take. The valuable experiences gained in his study of the yellow fever outbreak at Fort Columbus resulted in his being detailed in 1872 for service at New Orleans, La., and Fort Barancas, Florida—both in the so-called yellow-fever zone. At the Florida post he passed through two epidemics, in the second of which he himself contracted a severe attack of the disease.

The first of Dr. Sternberg's scientific publications was a clinical description of yellow fever as witnessed by him in the several epidemics through which he had passed. In 1878, while post surgeon at Fort Walla Walla, on the Pacific slope, he began investigations upon the value of commercial disinfectants—a line of work with which his name was conspicuously identified until the question was finally and satisfactorily settled. His experiments, begun at an army post, were continued in Washington, D. C., and at the Johns Hopkins University, where the importance of his researches led to his being awarded a "Fellowship by courtesy." He was at that time of the rank of Major, detailed for post duty at Baltimore. The expenses incidental to so costly an investigation were borne by the American Public Health Association, of which he was at one time president, and for years an active and influential member. The results of his investigations upon disinfectants won for him the "Lomb Prize" in 1886. The honor of having placed the whole question of disinfection on a scientific basis belongs conjointly to Sternberg and Koch. It was during his fundamental investigations upon the value of commercial disinfectants, made under a grant from the American Public Health Association, that I became his assistant, and it was from him that I received my first instruction in bacteriology. Were it not for fear of wandering too far afield, I would

relate some of my experiences while associated with this remarkable man. At that time the accurate, simple and logical methods of Koch were not available to workers in American laboratories. In fact there were almost no workers at all in pure bacteriology in this country, General Sternberg, to my knowledge, being the only one, and I can assure you that I appreciated my good fortune when the opportunity presented for me to serve as his assistant. Only those familiar with the exacting nature of a serious bacteriological research, conducted by the immature methods of that time, can appreciate the magnitude of his work on disinfection—and when it be made known that through all that investigation he was ably conducting the affairs of the military post to which he was detailed, one realizes to some extent the tireless energy and love of work with which he was endowed. Coincident with his studies of disinfectants were researches in other fields of bacteriology. In 1880 he discovered in the saliva a micrococcus to which he gave the name "Micrococcus pasteuri." As he had found it in his own saliva and in the saliva of many other normal human beings—and as he had demonstrated that its introduction into the tissues of certain animals resulted only in fatal septicemia—it is not astounding to find him surprised when Fraenkel declared pneumonia in man to be caused by an organism identical with "Micrococcus pasteuri;" which organism is now generally known as "pneumococcus." The literature of the time on the subject is somewhat confusing, due in large part to the limitations of and to the differences in available technic—but nevertheless, call the organism what we may, it was Dr. Sternberg who discovered it and described many of its peculiarities, though he failed to recognize its most important activities. In 1881 he proved conclusively that the so-called "Bacillus malariæ" of Klebs and Tomasi Crudelli, then attracting wide-spread attention, had nothing to do with the causation of malarial fever and in 1885 demonstrated for the first time in this country the living, motile Plasmodium malariæ first seen by Laveran in 1880, and subsequently proved to be the cause of malarial fever.

Of the many and varied problems with which Dr. Sternberg

was identified, probably none absorbed more of his energies than that concerning yellow fever. It would take more time than is allowed me to follow his studies of this disease in all their ramifications. We can, however, content ourselves with saying that through his individual effort, *i. e.*, investigations made by him personally—he closed once and for all time the question as to the likelihood of yellow fever being a disease of bacterial origin. Only those who were on the spot and saw the remarkable tenacity with which he followed out to its termination every promising lead, can realize the determination of a man whose only reward for an enormous expenditure of energy was negative results. But we must not underestimate the value of such results—remember, please, that at the time of which I write almost nothing was known of yellow fever beyond its clinical manifestations, and bacteriology was hailed as the science through which the riddle was to be solved. Errors in abundance were made and had to be corrected. Dr. Sternberg did this. Having exhausted the subject insofar as the available bacteriological, microscopic and inoculation methods would permit, Dr. Sternberg conceived the idea of experiments on human beings. Dr. Carlos Findlay, of Havana, had already made certain inconclusive investigations in the way of protective inoculations of man through the use of infected mosquitoes. The next step was the appointment of the U. S. Army Commission with Major Walter Reed at its head. The results of the activities of that commission in enlightening us upon the subject are too well known to require comment at this time. But the essential thing to remember is that the organization of the Yellow Fever Commission was General Sternberg's idea.

Not the least important of his many activities were those concerned with photomicrography, of which he was a master. He was the first in this country to reproduce by photography Bacillus tuberculosis discovered by Koch in 1882. In 1884, in his revision of his 1880 translation of Maignin's *Bacteriology*, he published a large group of photomicrographs of bacteria that were comparable in their excellence to many that have subse-

quently appeared, and superior to any in existence at that time. In 1892 appeared the first edition of his own book, *A Manual of Bacteriology,* and in 1896 a revision entitled a *Text-book of Bacteriology.* In addition he was the author of several other books on special topics, notably *Malaria and Malarial Diseases*: *Immunity, Protective Inoculation in Infective Diseases and Serum Therapy; Infection and Immunity, with Special Reference to the Prevention of Infectious Diseases.*"

He was a frequent writer of chapters for collaborative books, of articles for encyclopedias, and more than sixty special scientific papers stand to his credit.

During the time of his Surgeon-Generalship (1893 to 1902) he established the Army Medical School; established laboratories for scientific investigations at most of the important army posts; provided all new army hospitals with modern operating rooms and encouraged their use by surgeons in the service; he established the army tuberculosis hospital at Fort Bayard, New Mexico. Immediately after the declaration of war with Spain, within four days, to be exact, he issued a circular upon camp sanitation and dwelled upon the possible role of flies in disseminating typhoid fever. This warning was not heeded, with results well known to all of us.

He organized the board for the investigation of typhoid fever; composed of the late Major Walter Reed, the late Dr. E. O. Shakespeare, a Fellow of this College, and Dr. Victor C. Vaughan, of the University of Michigan. Upon his recommendation Drs. Vaughan and Shakespeare were commissioned as surgeons of volunteers. As said, he organized the Yellow Fever Commission, composed of Major Walter Reed, Drs. Carroll, Lazear and Agromonte. During the Spanish-American War he organized eight army hospitals at appropriate points and equipped two hospital ships and one hospital train. He organized the female nursing corps and the corps of dental surgeons in accordance with an act of Congress, passed at his suggestion, and recommended a large increase in the medical corps to correspond with the increase in the army made in 1901.

After his retirement from active service—necessitated by the age limit—he devoted his tireless energies to what he regarded as the duties of a citizen. He was affiliated in a conspicuous way with many organizations having to do with the welfare of the National Capital. He was President and Founder of the Washington Sanitary Improvement Company; of the Washington Sanitary Housing Company; President of the President's Homes Commission; President of the Citizens' Relief Association; President of the Washington Sanatorium Company; Director of the Starmont Sanatorium; Chairman of the Committee on the Prevention of Tuberculosis; member of the Committee on the International Tuberculosis Congress; President of the Board of Directors of Garfield Hospital; President of Board of Visitors of St. Elizabeth's Hospital, and Professor of Preventive Medicine in George Washington University. He held membership in many important societies. Besides being a Fellow of this College, he was a member and ex-President of the American Public Health Association; member and ex-President of the American Medical Association; member and ex-President of the Association of Military Surgeons; member and ex-President of the Philosophical Society of Washington; of the Biological Society of Washington; of the Cosmos Club; honorary member of the Association of American Physicians; of the New York Academy of Medicine; of the Epidemiological Society of London; of the Academy of Medicine of Rio de Janeiro; of the American Academy of Medicine, and of the French Society of Hygiene. The Honorary Doctorate of Laws was conferred upon him by the University of Michigan in 1894, and by Brown University in 1897.

Dr. Sternberg was what we are pleased to call a self-made man. His early environment was certainly not luxurious: A school-teacher at sixteen; a student of medicine on borrowed money at nineteen, all of which money he subsequently earned and returned to the lender; a surgeon in the United States Army at the age of twenty-three—speaks for individual capacity that bade well to carry its possessor far in the race for preferment—as his subsequent history has well shown to have been the case.

An incident during the course of my acquaintance with him often impressed me with his will and energy. At the age of fifty-five or thereabouts he knew nothing of the German language. It was at a time when the most important of our researches were emanating from the German laboratories. He knew French intimately, but that helped only in part. Was he discouraged? Not at all. With the aid of a tutor and by close application he acquired a trustworthy reading knowledge of German in less than two years.

Though I knew General Sternberg more or less intimately from 1884 until the date of his death, I never so fully appreciated the magnitude of his attainments, his services to mankind, or his devotion to duty as I did in the course of preparation of this minute. In the death of General Sternberg, this College loses one of its most distinguished fellows; American medicine, a pioneer of whose attainments it may be justly proud, and those who knew him well, a faithful, kindly friend.

MEMOIR OF WILLIAM L. RODMAN, M.D.[1]

By JAMES W. HOLLAND, M.D., Sc.D.

After the lapse of more than half of this year, memorable in history for its succession of startling events, we are still thrilled as we recall the emotion caused by the untimely death of our fellow-member, William L. Rodman. His riper years were set to large issues that concerned some of us very closely. His sudden release from earthly cares made a difference to not a few of our progressive councils.

Born in Frankfort, the capital of Kentucky, he was the son of a lawyer who for some years was Attorney-General of the State. He grew up in an ordered and cultured home, with a genial social environment of great political activity. Two of his relatives were noted physicians, that by their engaging manners, joined to real merit, rose to public offices of honor in our profession. Fruit from a twig of good stock grown in favorable conditions, both nature and nurture conspired to make possible his exceptional career. His preliminary education was acquired at a literary college in his native city that gave to its students the military training so much called for in this time of wars and rumors of war.

This college course made a sound basis for his subsequent success as military surgeon, teacher, and public speaker. His medical lectures and addresses in later life were characterized by a facile and dignified style set off with the grace and force of one whose native bent for oratory had been highly developed by practice.

Before joining the classes of Jefferson Medical College, he had pursued the customary apprenticeship by reading medicine with his cousin, Dr. William B. Rodman, a graduate of Jefferson Medical

[1] Read November 1, 1916.

College. While a student of medicine he enjoyed the instruction of such able men as the elder Gross, Jacob Da Costa and Theophilus Parvin. After graduation he seized upon the opportunity to serve as interne at the Jefferson Hospital, then operated under a staff that included Richard Levis, John Brinton, and the younger Gross. Seeking for a way of life, his mind was first attracted to the Army Medical Corps, of which he became a member, serving as assistant surgeon for nearly two years at Fort Sill, Indian Territory. In recent years this military episode proved to be the source of valuable special information, as well as of satisfaction, in that he had shared in that corporate loyalty to his country, which should be the essence of a soldier's life. There being little prospect of active service or promotion in those piping times of peace, he returned to civil life to marry the lady whose devotion to him and their children made of their home a model of domestic happiness.

Feeling assured that he could make his way where his ambition called him, in a larger theatre, he removed to Louisville, Ky., and while waiting for private patients, took up the duties of demonstrator and clinical assistant in surgery at the university there. In four years he made his mark and was elected professor of surgery in the rival Kentucky School of Medicine. Five years afterward, his fame as a lecturer and operator having spread far afield, he received practical recognition by his election to the chairs of surgery in the Medico-Chirurgical College, and the Woman's Medical College, both of Philadelphia. His chief contributions to literature were the oration in surgery on "Gastric Ulcer," a paper on "Cancer of the Breast," read before the British Medical Association, his well-known monograph on *Diseases of the Mammary Gland*, and sundry chapters contributed to three different systems of surgery.

Something of an adept at political management, his extensive acquaintance made in his several migrations and as teacher in four medical colleges directed his energies to a wider field. From the contacts of his army connection and his southwestern sojourn in his early manhood, he had imbibed the free spirit of the West and a broad Americanism that took shape in various efforts with a national scope. For many years he played an active part in the

affairs of the American Medical Association; presiding over the Section on Surgery at one meeting, and at another delivering the oration on surgery; member of the house of delegates, of the board of trustees and finally president.

For a long period he was one of the Committee on National Legislation and again chairman of the Committee on Reciprocity. As a final result of these labors he became convinced that the best solution for the problems with which he had to deal (such as the right to practice not limited by State lines) lay in the plan for a voluntary National licensing board which should contain representatives of the Medical Corps of the Army, the Navy and the Public Health Service; the Federation of State Examining Boards, and the profession at large. Its standards were to be the highest then obtaining anywhere in the Union, its examination to be more thorough and exacting than any then officially approved.

With his usual sagacity he recognized this as his mission when the call came, and the occasion offered. To the attainment of this end he brought an extraordinary mental energy and his strong personality. They gave him unique standing and attracted the definite adhesion of cautious but sympathetic neutrals when he appeared as advocate before the many official boards concerned. The natural jealousies and rivalries of established licensing boards fortified by the political doctrine of State rights, created a critical atmosphere and a situation in which all the chances were against him.

Having worked out his problem in advance and having conceived a practicable scheme, he secured financial aid for it from the Carnegie Foundation, adapted it to meet reasonable objections, put it on a permanent basis, and at last justified it by the approval of the most influential medical organizations in this country. A decided impetus was given to the movement by his election as President of the American Medical Association. Having got his hook in the nose of the leviathan, he used his control to further his progressive policies as defined in his presidential address at San Francisco. He helped materially to strengthen the preliminary requirement to the practice of medicine demanded by the colleges and the State examining boards.

Our international complications induced the last Congress to set about preparing for war. As an ex-army surgeon and as titular representative of the medical profession, he was consulted by the military committees of that body in the later months of his life. His opinion was sought upon the best plan of reorganization of the medical department of the army and on general measures for medical military preparedness. He had cherished ideas concerning medical education in Philadelphia which came to partial fruition in the merger of the Medico-Chirurgical College with the graduate department of the University.

Without counting the cost, he spent the utmost powers of his mind and the last reserves of his bodily strength in pushing his enterprises for medical public institutions before the Council of Medical Education, the Confederation of State Examining Boards and divers medical meetings in all parts of the country. Meanwhile he had to sustain the anxieties and fatigues of surgical practice and his professional duties. A constitution of iron had been denied him by nature, but even that, had he possessed it, would have been sorely tried by these multifarious and wearing exertions. No wonder that exposure to cold and infection found him lacking in the physical stamina adequate for resistance. To the last his mind remained potent and creative, but his overtaxed strength gave way, and after a few days of illness the end came. Although his campaigns did not always work out as he had planned, looking back over his many victories and few defeats, one must conclude that events had been so often shaped to please him that he might well have been content with his record. By his achievements he left his country and his chosen calling other and better than he had found them. By assisting in the betterment of our professional standards and creating a National body for that purpose which should serve as exemplar to the States he made a notable contribution to the construction of society.

It is a sombre privilege to echo here the thought he once expressed to the writer of this memoir, "The National Board of Medical Examiners will be my monument."

THE ANNUAL ADDRESS OF THE PRESIDENT[1]

By JAMES CORNELIUS WILSON, A.M., M.D.

For the third time it has become my duty to communicate to you from the chair a brief review of our affairs at the close of a College year.

There were during the twelvemonth just ended nine stated meetings at which the average attendance was $50\frac{2}{3}$, a falling off of 6 from the mean of the preceding year.

One special meeting was held at the call of the President to listen to an Address by Doctor Victor Clarence Vaughn, of Ann Arbor, Michigan, entitled: "A Study of Camp and Military Hygiene, its Sanitation and its Relation to War Mortality."

There have been no memoirs of deceased Fellows read before the College during the past year.

The scientific business has continued to advance the laudable purposes which the Founders of the College had in view as set forth in the Charter which was Granted them and Enacted into a law, at Philadelphia, on Thursday, the twenty-sixth day of March, in the year of our Lord one thousand seven hundred and eighty-nine by the Representatives of the Freemen of the Commonwealth of Pennsylvania, in General Assembly met, and by the Authority of the same, as duly attested by Richard Peters, Speaker, and Peter Zachary Lloyd, Clerk.

Twenty-two papers were read at the stated meetings, mainly by the Fellows of the College; but seven gentlemen not Fellows took part in their presentation by special invitation. Six of these communications were illustrated by means of the projectoscope.

[1] Read January 5, 1916.

The discussion which followed was in most instances general and added greatly to the interest of the meetings. This year the scientific business has more than maintained the traditions of the College in the consideration of the results of the most advanced thought and rigid investigation in the several departments of medicine. In fact, a majority of the communications concerned matters of vital importance to the Art of Medicine which are now engaging the attention alike of laboratory workers and clinicians in a world-wide study to ascertain their usefulness and determine their limitations in actual practice—a field of work explicitly set forth in the application of the Founders for the charter.

The Sections of the College have been carried on with increasing activity and success and the attendance has been larger than in former years. It is earnestly to be hoped that the plan to publish the work of these important departments in the annual TRANSACTIONS will be realized in the near future.

The Committee on Publication again deserves the thanks of the College for the efficient manner in which it has performed its laborious duties. Volume XXXVI of the Third Series of the Transactions was published early in 1915. It contains cxiv + 388 pages, 107 less than that of last year. Four important memorial addresses commemorating the life and labors of deceased Fellows, all of them of great interest and historical value, occupy 56 pages; 58 pages contain the lists of officers, members of the Committees and the Fellows, while the remaining 388 pages are devoted to the Address of the President and the Scientific Business presented at the meetings. The fact that the twenty-two communications upon medical subjects during this year are presented upon 107 fewer pages than the nineteen papers of the previous year indicates in a general way a growing tendency to conciseness and directness of statement and increased care in the preparation of this part of our work, which is greatly to be commended. For the first time the back of the title page of the TRANSACTIONS bears the statement that "This volume is published in part by the income from the Francis Houston Wyeth Fund."

The Library Committee has presented a report that is most

satisfactory to the Fellows of the College. It shows a steady growth and increasing usefulness of this department of our organization, which more perhaps than any other of our activities justifies our established position as an Institution of Learning. This report is largely statistical and will, in accordance with the custom of the College, be printed as a separate document. I desire, however, to emphasize certain matters of importance, to which it calls attention.

The total number of books, including bound volumes and 10,287 unbound "Reports" and TRANSACTIONS, is 107,782, an increase of 2242. There are in addition to this number 12,949 unbound "Theses" and "Dissertations" and 90,870 unbound "Pamphlets." From all sources we received during the year, 3724 volumes, 12,522 pamphlets and 20,790 numbers of various periodicals; very many of these were duplicates. The individual donors numbered 461. The number of new publications added to the Library during the year was 574. Of these 52 were written or edited by Fellows of the College. The visits paid to the Library during 1915 numbered 8270; of these visits 3263 were made by Fellows of the College. In this connection it is well to remind you that ours is in a certain sense a free library. The general as well as the professional public are welcome to consult any of the books in the Reading Room, or the periodicals in the Journal Room. Many persons, not physicians, but interested in medical history or the collateral sciences and an occasional literary person avail themselves of this opportunity. The greater number of visitors to the Library are, however, members of the medical profession. The Library has been kept open two evenings each week and on the six minor legal holidays. The number of books consulted in the Library was 25,770; the number taken out 4799. The "Study Rooms" have been in constant demand during the year, the average number of volumes called for and retained in use in these rooms being 175.

During the past year the Library Endowment Funds have been increased by the Louis A. Duhring Book Fund $5000, and the Louis A. Duhring bequest for the general purposes of the Library,

$181,122.02. The Library Endowment Funds now aggregate $292,318.85.

The following is a list of the rare medical books and works of special interest received during the past year:

Incunabula

[Total number of incunabula at this date, 187]

Aegidius Columna. [De regimine principum.] Romae, Plannck, 1482. F°. [Hain no. 108.]

Fund for Rare Books.

Albertus Magnus. [Physicorum sive de physico auditu libri octo.] Venetiis, de Forlivio, 1488. F°. [Hain no. 518.]

Fund for Rare Books.

Aristotelis. [Opera graece.] Venetiis, Aldus, 1495–98. 5 vols. F°. [Hain no. 1657.]

A superb copy of the first edition of the work of Aristotle, printed in Greek characters. The value of this work is increased by the fact that it was one of the earliest examples of books printed entirely in Greek characters. Dr. Fielding H. Garrison says: "The greatest scientific name after Hippocrates is that of the 'Master of those who knew,' the Asclepiad Aristotle (384–322 B.C.) who gave to medicine the beginnings of zoölogy, comparative anatomy and embryology, and the use of formal logic as an instrument of precision."

Presented by Henry Reed Hatfield, Esq.

Bergomensis, J. P. [Supplementum chronicarum.] [Venetiis], Bernardinus de Banaliis, 1483. F°. [Hain no. 2805.]

Fund for Rare Books.

Ficinus Florentinus, Marsilius. De triplici vita. [Parisiis, Wolf, ca. 1492.] 16°. [Copinger no. 2497.]

Fund for Rare Books.

Firmicus Maternus, Julius. De nativitatibus. Venetiis, Simon de Bevilauqua, 1497. F°. [Hain no. 7121 bis.]

This is a first edition with a good example of a Xylographic Gothic title, and a woodcut printer's device on the last page. This work is quite rare, and, while not medical as a whole, contains much pertaining to medicine.

Presented by Drs. Charles W. Burr and Edward B. Krumbhaar.

Jung, Ambrosius. [Tractatus perutilis de pestilentia ex diversis auctoribus congregatus.] Augsburg, Schonsperger, 1494. 4°. [Hain no. 9473.]

A rare German incunabulum.

Presented by Drs. James M. Anders, Thomas G. Ashton, George M. Boyd, Charles W. Burr, Barton Cooke Hirst, David Riesman, Joseph Sailer, James C. Wilson, George Woodward and Sir William Osler, Bart.

Lactantius Firmianus, L. C. [Opera.] [Venice], 1471. F°. [Hain no. 9809.]

Fund for Rare Books.

Rodericus Sanctius. Bishop of Zamora. [Speculum vitae humanae.] Augsburg, Zainer, 1471. F°. [Hain no. 13940.]

A very rare and beautiful specimen from the first press at Augsburg. This is the earliest dated book in the Library of the College, and, while not a medical work, contains a number of passages relating to medical subjects.

Presented by Drs. George E. de Schweinitz and Richard H. Harte.

Serapion [Joan] the younger. [Liber Serapionis aggregatus in medicinis simplicibus.] Venetiis, Raynaldus de Novimagio, 1479. F°. [Hain no. 14692.]

Fund for Rare Books.

Wirecker, Nigellus. [Speculum stultorum.] [Leipzig, Kachelosen, ca. 1494.] 8°. [Hain no. 16217.]

Fund for Rare Books.

Works of Special Interest

Bayle, Pierre. Dictionnaire historique et critique. 3 ed. Rotterdam, Bohm, 1720. 4 vols. F°.

This bibliographical dictionary is considered Bayle's masterpiece. It was first issued in 1695–97 and has become quite rare.

Presented by Dr. Richard H. Harte.

Brandt, Sebastien. [Stultafera navis.] [Basel, Joh. Bergmann de Olpe, 1505.] 8°.

This book is a Latin version, by Jacobus Locher, of Sebastien Brandt's famous "Das Narrenschiff" (Ship of Fools), which first appeared in 1494. This copy lacks the title page and several pages of text.

Presented by Dr. Edward B. Krumbhaar.

Cardanus, Hieronymus. Metaposcopia. Lutitiae Parisiorum, Jolly, 1658. F°.

This book is quite rare, not having been included in the collected editions of the works of Cardanus.

Presented by Dr. John K. Mitchell.

Carmona, J. Tractatus de peste ac febribus cum particularis. Hispali, Maldonado, [1582]. 24°.

By Purchase.

Clark, James. Treatise on the yellow fever as it appeared in the island of Dominica in the years 1793–4–5–6. London, Murray, 1797.

By Purchase.

Goodall, Charles. The Royal College of Physicians of London. London, Kettilby, 1684. 8°.

Presented by Sir William Osler, Bart.

Ingram, Dale. Historical account of the several plagues that have appeared in the world since 1346. London, Baldwin, 1755. 8°.

Fund for Rare Books.

Mercado, Luiz. Libellus de essentia causis, signis, et curatione febris malignae. Pinciae, D. F. à Corduba, 1574. 24°.

By Purchase.

Pirogoff, N. I. Anatome topographica. Petropoli, Trey, 1859. 5 vols. F°.

Pirogoff, the greatest Russian surgeon of his time, is remembered today by his operation of amputation of the foot—"Pirogoff's amputation;" but his Anatomy, a monumental work of great value, in which frozen sections were first used in the process of illustration, seems almost unknown to the present generation. Complete copies are quite rare.

Fund for Rare Books.

Priestley, Joseph. Directions for impregnating water with fixed air. London, Johnson, 1772.

Presented by Sir William Osler, Bart.

Röslin, Eucharius. Der swangern Frauwen und Heebamen Rosengarten. Argentine, Flach, 1513. 4°.

First edition of the first work printed on obstetrics. This work is based on a manuscript by Moschion, a writer of the second century. The "Rosengarten" was translated into English by Thomas Raynald, under the title "The Birth of Mankynd." The binding is an excellent example of early sixteenth century German work.

Presented by Drs. James M. Anders, Thomas G. Ashton, George M. Boyd, Charles W. Burr, Barton Cooke Hirst, David Riesman, Joseph Sailer, James C. Wilson, George Woodward and Sir William Osler, Bart.

Sophia, Marsiliis de Sancta. Opus aureum ac preclarum, de recent memoria in luces traditum. Lugduni, 1517. 12°.

Fund for Rare Books.

Other Interesting Additions

Aesculapius. Photograph of a statue.

Presented by Dr. James V. Ingham.

American Association for the Advancement of Science. Silver badge, 1914.

Presented by Dr. W. W. Keen.

Cow-Pock, or the Wonderful Effects of Inoculation. Engraving.

Presented by Miss Mary Lee.

Duhring, Louis A. Portrait in oils by H. H. Breckenridge.

Ordered by Resolution of the College.

La Roche, Réné. Portrait in oils. (Artist unknown.)

Presented by J. Percy Keating, Esq.

Purgation-Calendar, 1453. Facsimile of original, printed in Gutenberg types, in the Bibliothèque Nationale, Paris.

Presented by Dr. Fielding H. Garrison.

Vesalius, Andreas. Engraving of the painting "Vesalius Demonstrating."

In Exchange.

We have also received during the year many interesting and more or less valuable autograph letters.

Through the generosity of Dr. Francis R. Packard, the Library Committee have been enabled to have additional steel cases erected in the "Packard Room" to give much needed space for our growing collection of bound manuscripts.

The Committee on the Mütter Museum report the frequent use of the specimens to illustrate lectures delivered and papers read before the College and at meetings of various societies held in the College Building. The number of visits to the Museum has greatly increased, especially on Wednesday and Friday evenings. New cases have been placed along the railing of the balcony, greatly facilitating the study of the specimens and improving the appearance of the hall. Further plans looking to these ends are to be carried out in the near future. The lecture for 1915 by Professor Rudolph Matas, of New Orleans, upon

"The Principles Governing the Surgical Treatment of Peripheral Aneurisms," announced for December 17th, was postponed on account of serious illness in the family of the lecturer.

The financial condition of the Mütter foundation is most satisfactory.

The Hall Committee has administered its exacting and responsible duties with economy and efficiency. There has been some delay in carrying into effect contemplated changes in the lighting system in order to be sure of obtaining improvements that will be permanently satisfactory. The Sinkler Memorial Garden has been during the greater part of the year a source of great pleasure to the Fellows of the College and others and the general condition of the Hall has been in every respect satisfactory.

The Committee on the Directory for Nurses report a year of success is every particular. I must again remind the Fellows that the Directory of the College is administered in the interest of the medical profession and the nurses alike, but chiefly in the interest of the public, and that its methods and efficiency fully justify its asking for the full support of these three groups of citizens.

The Committee on Finance, of which the Treasurer is an active member, has not only discharged its routine duties with remarkable faithfulness and skill but has also closely watched our financial interests in every particular. These interests have during the year just ended been of unusual importance, owing to the fact that a large number of miscellaneous securities have come into the possession of the College in the settlement of the Duhring Estate. The Treasurer has been untiring in his efforts to administer the duties of his office and his labors have been crowned with success. The management of this department of the College involves increased responsibility as our resources grow. We must never overlook the fact that such income as comes to us is to be spent in increasing our usefulness to the community, and that in regard to our expenditures we are in all cases to exercise that reasonable prudence which is proper to an institution of learning.

The successful work of the Committee on Scientific Business has been laid before you in the early portion of my remarks. The efficiency of this important Committee must be ascribed to its ability to secure communications of a very high order for our meetings and to keep the Fellows interested and alert in this essential field of our activity. This Committee has again been able to conduct its work without expense to the College.

The Committee in Public Health and Preventive Medicine has manifested a revival of interest on the part of the College in matters regarded as most important in our early history but which for a period had attracted less attention than was proper. During the year it arranged a "Symposium on National Hygiene," which was held at the stated meeting of April 7, and in response to public demand obtained for the Director of Public Health and Charities the opinion of the College in regard to the proper disposition of cases of tuberculosis constituting a menace to the health of those surrounding them; and forwarded to the Governor of the Commonwealth of Pennsylvania a copy of a resolution, unanimously passed by the College, in opposition to a bill inimical to housing and sanitary conditions in Philadelphia. At the last meeting of the College this Committee submitted to the College a resolution in regard to the treatment of victims of various drug-habits, offered in order to obtain the sense of the College in reference to a request from the Advisory Committee of the Misdemeanants Court. In view of the importance of the matter the College referred the matter to the Council.

To all these Committees and their Chairmen the College desires to express its sense of appreciation of their labors and its warm thanks.

At the December Meeting the College adopted the Resolutions recommended by the Council, presented by Dr. C. H. Frazier, Chairman of the Sub-Committee in Efficient Laws on Insanity: "That a separate State Board be appointed charged with the sole duty of seeing to it that the provisions of the various Acts of Assembly relating to the insane, defectives, and epileptics, respectively, in this State are carried out," etc.

At the same meeting the College adopted the Preamble and Resolutions presented to the Council by the Committee on Public Health and Preventive Medicine, and favorably recommended, in regard to the prevention of the plague and other diseases caused by internal parasites.

Dr. Packard, as Chairman of the Committee appointed to gather information concerning the Fellows who served as officers in the Army, Navy, and Public Health and Marine Hospital Service of the United States during the time of the Civil War, presented two volumes for preservation in the Library, one containing a list of the names of the Fellows with such data as could be obtained, the second containing the maps, letters, and photographs relating to the Hospitals at Gettysburg, received from Dr. S. Weir Mitchell and Colonel Nicholson.

The report was accepted and the Committee discharged. The thanks of the College were extended to Dr. Packard for these volumes and the admirable manner in which the papers had been mounted and bound for preservation.

At the same meeting two important amendments to the By-Laws were adopted; the first reducing the entrance fee and annual dues; the second creating the Committee on the Weir Mitchell Oration. To these changes I alluded in my address of last year.

At the June meeting the President announced that he had issued a circular letter to a number of prominent educational and scientific institutions stating that "the College of Physicians of Philadelphia has in preparation a list of such bodies with which it desires the honor and privilege of exchanging the courtesy of representation by official delegates at important public ceremonials of general interest," and read a number of invitations received by him officially as President of the College.

At the same meeting the President announced the receipt of five thousand dollars ($5000), a bequest under the will of Elizabeth S. Shippen, deceased; also the receipt of securities and cash in the matter of the settlement of the estate of Louis A. Duhring.

At the October meeting the President announced the appoint-

ment by the Committee on the Weir Mitchell Oration, of Dr. George E. de Schweinitz to deliver the first oration in the Autumn of 1916.

The Alvarenga Prize for 1915 was awarded to Dr. J. E. Sweet, of Philadelphia, for his essay entitled, "The Surgery of the Pancreas."

In the course of the year just ended the College has lost by death six Fellows:

Dr. Richard Flavel Woods, elected a Fellow in 1901, died on February 10, 1915. Dr. Woods was educated at Princeton College, receiving his A.B. degree in 1892, and that of A.M. three years later. He entered upon his medical studies at the University of Pennsylvania in 1892, and after graduation in due course served as resident physician in the Pennsylvania Hospital and later engaged in dispensary work in the Pennsylvania and Presbyterian Hospitals. He subsequently became interested in gynecology and was at the time of his death assistant surgeon to the Gynecean Hospital and gynecologist to the Presbyterian Hospital. Upon the death of his father he succeeded to a large private practice to which he chiefly devoted himself until his health began to fail early in the year 1913. Gifted with unusual literary ability, he was an occasional and most acceptable contributor to current magazine literature and to the medical journals. He was, however, most useful to his day and generation as a medical practitioner of high type, skilful, consciencious and devoted.

Dr. Thomas Biddle, elected a Fellow in 1884, died on February 19, 1915. The son of Jonathan Williams and Emily Meigs Biddle, Thomas Biddle was born in Philadelphia in 1853. He was graduated in medicine at the University of Pennsylvania in 1876 and practised his profession in Philadelphia for some years. For a time also he was interested in public affairs and served with probity and wisdom on the Board of Guardians of the Poor. He early developed an unusual fondness for natural history and became known throughout the world as a naturalist. It was possible for him to devote his abilities to scientific studies and he became closely identified with the Philadelphia Zoölogical

Society and the Academy of Natural Sciences. In both of these organizations he was until the time of his death an active spirit and efficient officer. He presented many rare and important specimens to these institutions, together with valuable books and photographs. His virile intellect, his generosity, unselfishness and kindliness of manner endeared him to a wide circle of friends.

Dr. Fred Harlen Klaer, elected a Fellow in 1910, died February 27, 1915. Dr. Klaer was graduated at Amherst College in 1900 and entered at once upon the study of medicine at the University of Pennsylvania, receiving his M.A. degree in 1904. He then became resident physician to the Hospital of the latter institution and was appointed at the close of his service chief resident physician, a position which he held for some years, resigning it in order to enter the medical dispensary. He was subsequently placed in charge of this service, which under his efficient supervision became in a few years one of the best organized medical dispensaries in the United States. During this period he was connected with the Medical School of the University, first as an assistant; subsequently as medical instructor, a position which he held at the time of his death. He was active among the younger men in the medical life of Philadelphia, and served for some years as recorder of the Pathological Society. He died of tuberculosis of the meningeal type, the victim of an infection contracted in the discharge of professional duty.

Dr. Thomas Hewson Bradford, elected a Fellow in 1884, died June 25, 1915. Dr. Bradford was graduated at the Jefferson Medical College in 1874. His early interests were in gynecology as a specialty, and he served several years as physician to this department in the out-patient service of the Pennsylvania Hospital. He did excellent work also in the dispensary of the Children's Hospital. Meanwhile he acquired a large and important private practice and established a high reputation as a practitioner. After several years he became deeply interested in the medical considerations of life insurance and gave up his general professional work to accept the appointment as medical director in two important companies, positions which he continued to

hold until his death. Dr. Bradford was led by hereditary inclination and a general fondness for local history to devote much of his leisure to the study of the early conditions of the Commonwealth of Pennsylvania and was for a long time an officer in the Society of the Colonial Wars. To high intelligence and force of character he added a very affable disposition and courteous manners, traits which endeared him to many friends.

Dr. John Bacon, elected a Fellow in 1903, died August 2, 1915. A graduate of Haverford College, Dr. Bacon studied medicine at the University of Pennsylvania, receiving his degree of M.D. in the year 1889. After graduation he served as resident physician in the University Hospital and later at the Johns Hopkins Hospital in Baltimore. He was for two years physician to the Eastern Penitentiary. For the succeeding period of his life he devoted himself to the general practise of his profession at Torresdale, occupying the enviable position of family physician throughout the countryside. Notwithstanding the arduous duties of a large country practice, Dr. Bacon took a warm interest in medicine as an organized profession, and was a member of the American Medical Association, the Philadelphia County Medical Society, and a frequent attendant at the meetings of this College.

Dr. Albert Roscoe Moulton, elected a Fellow in 1904, died August 16, 1915. Graduated in medicine at the Medical School of Maine in 1876, he early devoted himself to nervous and mental diseases as a specialty, and secured appointments which enabled him to pursue his studies in this field of medicine. He was for many years senior assistant physician to the Pennsylvania Hospital for the Insane; later physician to the Out-Patient Department for Nervous and Mental Diseases at the Pennsylvania Hospital, and professor of mental diseases in the Maine Medical School (Bowdoin College). Dr. Moulton's long experience in psychiatry and his unusual temperamental qualifications for this branch of medicine enabled him to render efficient and helpful service to a large proportion of the patients in every walk of life who came under his care in institutional work.

Of Associate Fellows the College has lost during the year two.

Dr. Charles J. Finlay died at his home in Havana August 20, 1915, and Dr. George M. Sternberg died at Washington, D. C., November 3, 1915.

Ten Fellows have resigned: three have forfeited their Fellowship, and two Fellows, deserving such distinction, have been relieved from the payment of the annual contribution.

The roll of the College on December 1, 1915 shows:

Fellows		462
Associate Fellows:		
American	19	
Foreign	14	
	—	33
Corresponding Members		3
		498

It is of interest to consider the place that the College of Physicians has occupied in the medical life of our city. Organized in 1787, two years before its charter was granted, it has now been in continuous active existence for more than a century and a quarter. For over fifty years during which Philadelphia established and maintained, as it continues to maintain, its reputation as the medical centre of the country, with successful medical schools and great hospitals, it was the only society composed of graduates of some years' standing and experience coming together for mutual assistance and the general medical service of the community.

The American Medical Association was formed in 1847, with an organization of the most complete kind, comprising in its membership State and County societies and including every section of the country. This epoch-making event marked the beginning of medicine as an organized profession in America. What the significance of the movement was is evident in the solidarity of the profession in our times and its splendid service to the public. In 1849 the Medical Society of the County of Philadelphia came into existence. That it should be numerically

great and correspondingly influential was a foregone conclusion. Never was it so powerful and useful as today. Every physician of good standing is elegible for membership and should belong to it.

There are thus two general medical societies in the community existing side by side, each doing the same work but doing it in different ways. There are many other societies, perhaps too many, devoted to the specialties and various particular interests. But the members of all these are also, with few exceptions, members of the County Society. This is especially true of the College of Physicians. Its Fellows are, with scarcely an exception, members of the County Society. The Fellowship of the College is, indeed must be, in the nature of things, recruited from the sister society. In fact, we do more than take our Fellows; we take our Officers from the same source.

The College has had 20 presidents. Since 1849 it has had 13, and 5 of these had previously served as presidents of the County Society. The latter has had 54 presidents, of whom 49 have been Fellows of the College and 5 its president. It is thus seen that we not only have the same interests and do the same work, but that we are also largely made up of the same men. We therefore work in close harmony. They give us their best men and when they have become familiar with our history and customs, our aims and purposes and have well and faithfully served the College, we elect from among them as opportunity occurs, our presiding officer.

The period of three years during which it has been my agreeable duty to act as your executive officer has been a period of singular good fortune to the College. Our friends have been very generous to us. The list of incunabula, rare books, pictures and oil portraits they have given us is a long and most interesting one. The Library, notwithstanding the almost complete arrest of scientific publications and medical journals in the belligerent countries, has made a reasonable increase. Its committee has been able to lay up funds for the purchase of valuable and rare books, which will appear in the market upon the coming of peace

and the resumption of commerce. It is peculiarly fortunate in the recent accessions to its special funds and in a large endowment for its general purposes. Our Hall and its beautiful garden are in excellent condition. The administration is of the best training and experience and highly efficient. The committees have known their duty and faithfully performed it. We have been the recipient of large gifts of money from the living and by bequest.

But more than all this, the scientific work and the service to the profession and the public which constitute the purpose of our corporate existence have been maintained at the high level of our best traditions.

We have had losses:

> "Time takes them home that we loved, fair names and famous,"

but the quick hand catches the torch from the hand that falters, and as the beloved Fellow falls by the way, the ranks are filled and the College moves on in its course of usefulness and power.

OPERATION FOR REMOVING THE GALL-BLADDER[1]

By JOHN B. DEAVER, M.D.

The type of operation performed in the German Hospital Clinic for the removal of the gall-bladder, and which I believe is as simple as any, enabling one to explore the common duct throughout its entirety with ease and at the same time to control the bleeding from the gall-bladder bed, is the following:

With the abdomen open, the gall-bladder and the right free border of the gastrohepatic omentum freed of adhesions (not that there are adhesions in all cases), this region is thoroughly walled off, when with retractors the assistant keeps the wound wide open. The edge of the liver and the fundus of the gall-bladder are grasped with the left hand, carrying a piece of moist gauze, pulled downward, outward, and upward, which makes taut the cystic duct and the free border of the gastrohepatic omentum (Fig. 1).

When the liver is adherent to the parietal peritoneum to the degree that it would not be wise to attempt severing the adhesions, it will not be possible to dislocate it in the above manner. In the presence of a diverticulum (a dilatation of the gall-bladder at its junction with the cystic duct, sometimes called the pelvis), the diverticulum conceals the upper portion of the gastrohepatic omentum and is frequently adherent to it. In either of the above conditions the cystic duct and the free border of the gastrohepatic omentum, between the layers of which lies the duct, are made prominent by grasping either the gall-bladder low down or the

[1] Read January 5, 1916.

FIG. 1.—Liver dislocated, gall-bladder exposed.

FIG. 2.—Hemostatic forceps grasping infundibulum of gall-bladder, right free border of gastrohepatic omentum with cystic duct made taut.

FIG. 3.—Cystic duct exposed, hemostatic forceps in position.

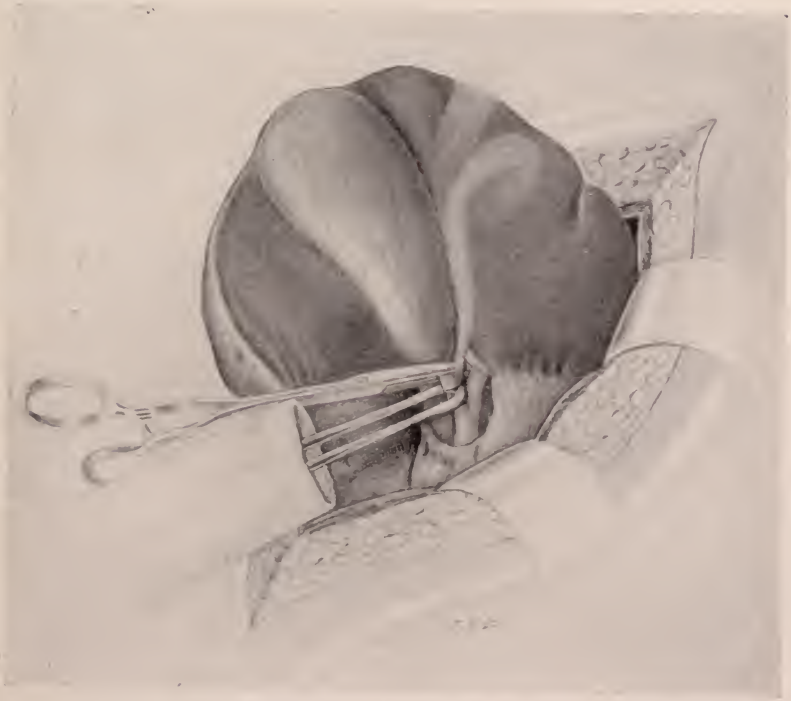

FIG. 4.—Cystic duct divided with cautery.

diverticulum with a long pair of curved forceps and making traction (Fig. 2).

When the diverticulum is adherent to the free border of the gastrohepatic omentum, it must be freed before effective traction can be made upon the cystic duct. Unless this be carefully done, the common duct may be injured, as occurred in one of the writer's cases. A small incision is made through the upper part of the free

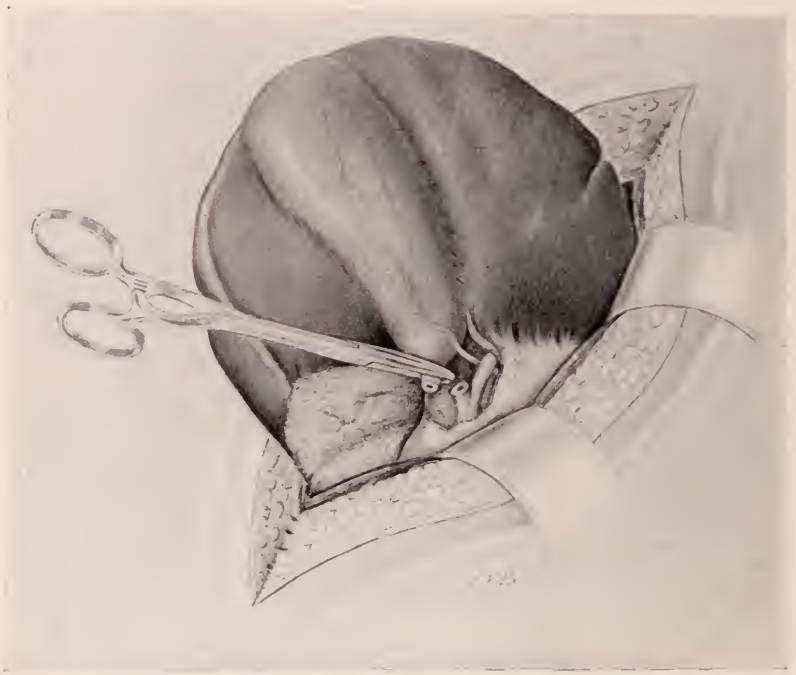

Fig. 5.—Orifice of cystic duct, cystic artery and hepatic duct exposed.

border of the gastrohepatic omentum and the cystic duct exposed (Fig. 3). The cystic duct at its junction with the gall-bladder is clamped with a long pair of hemostatic forceps and cut across distal to the forceps with the actual cautery (Fig. 4). A small piece of gauze is placed beneath the border of the gastrohepatic omentum to take up the bile that escapes when the cystic duct is divided (Fig. 5). A small, followed by a larger, probe is passed

into the stump of the open cystic duct and carried through the common duct into the duodenum (Fig. 6), in this way determining whether the duct and the papilla of Vater are patulous. Before the probe is withdrawn from the common duct, the duct is palpated by grasping the descending duodenum and head of the pancreas between the fingers and thumb of the free hand, when it

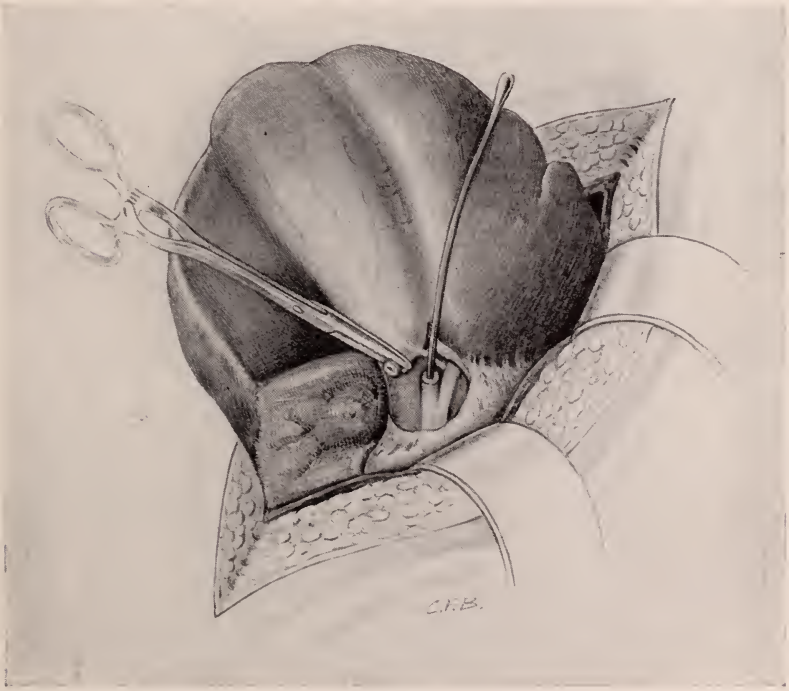

FIG. 6.—Probe introduced into orifice of cystic duct.

is definitely settled whether the common duct contains a stone or stones. Further, by this means, if the duct contains a stone which is not detected by the probe and yet the probe has passed into the duodenum, through the wall of which the end of the probe can be felt, the stone will be palpated, it can also be determined if there be obstruction outside of the duct. Next, the cystic artery, lying above and to the inner side of the cystic duct, is clamped and cut.

In a small percentage of cases the cystic artery lies below and to the outer side of the cystic duct. If it is not necessary to drain the common duct through the stump of the cystic, the cystic duct is ligated and next the cystic artery (Figs. 7 and 8).

The next step is the separation of the gall-bladder from the liver. This dissection is made from below upward, the gall-bladder

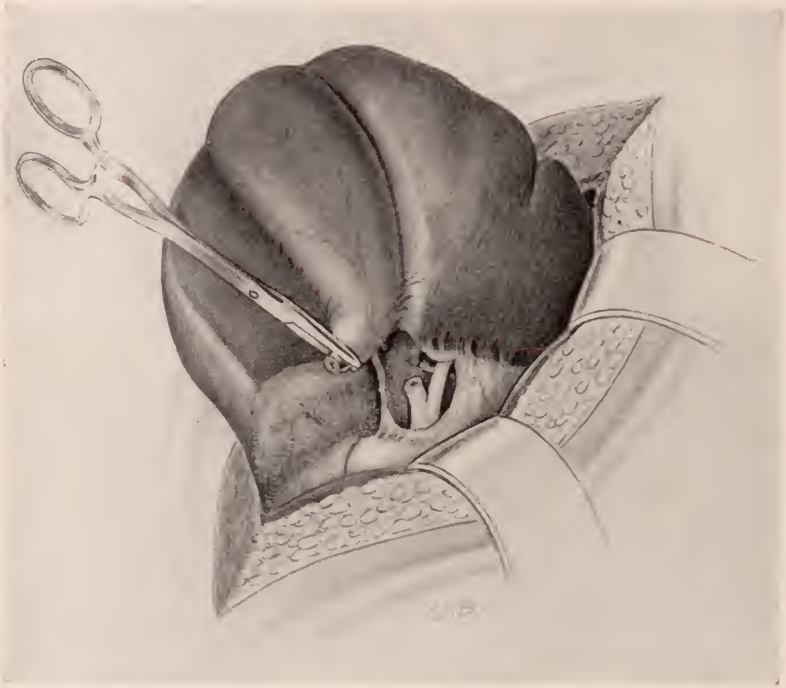

Fig. 7.—Cystic duct divided, hemostatic forceps upon neck of gall-bladder and orifice of cystic duct exposed.

is freed and turned upward and outward, and the gall-bladder bed closed by carrying a continuous catgut suture through the liver substance forming the sides and floor of the gall-bladder bed (Fig. 9). The free end of the suture is left long and tied to the portion of the suture the needle carries through the liver substance. The tie is made on the upper side of the line of apposition of the

walls of the gall-bladder bed. It will be seen that as the gall-bladder is removed step by step the suture is passed and tied, so that when the gall-bladder is entirely removed the gall-bladder bed is completely closed; by this means the procedure is made a bloodless one (Fig. 10). I regard this far preferable to packing the gall-bladder bed with gauze or placing a cigarette drain therein

FIG. 8.—Removal of gall-bladder, cystic duct and artery ligated.

and retaining it by suture. In a small percentage of cases in which the walls of the gall-bladder are not extensively infiltrated, it will suffice to incise the serosa in the long axis of the organ upon either side sufficiently far from where it is attached to the liver to cover the gall-bladder bed when the edges are apposed by a continuous catgut suture. The lateral flaps of the serosa are reflected and the gall-bladder removed from below upward, leaving enough

of the fibrous coat so as not to trespass upon the liver. There are a few instances when a gall-bladder can, to better advantage and with greater safety, be removed from above downward, and mencing the dissection at the fundus.

The next step is suturing the divided layers of the gastrohepatic omentum, but not covering in the stump of the cystic duct. The

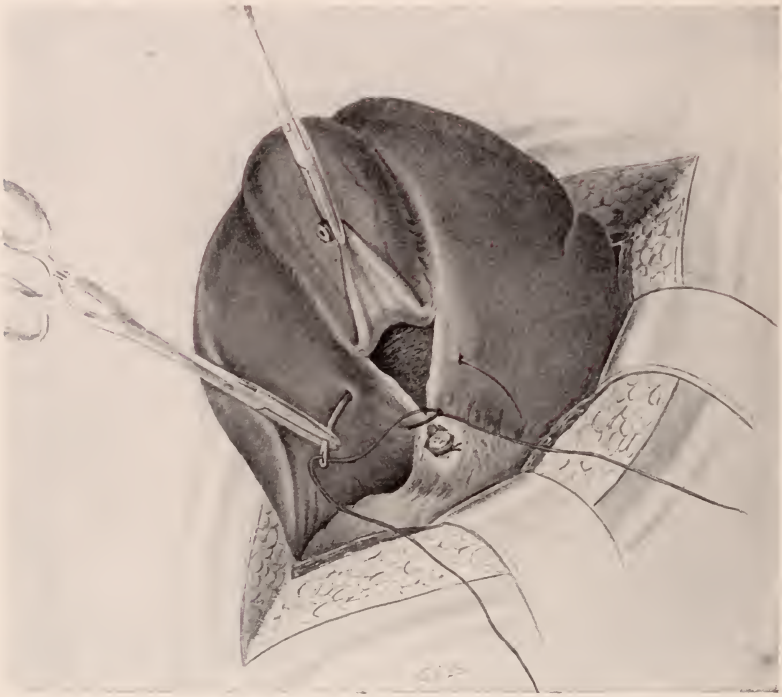

Fig. 9.—Commencing suture of gall-bladder bed

final step is the placing of a small rubber tube down to and just beyond the free border of the gastrohepatic omentum, which is retained for four or five days, so that in the event of the ligature on the cystic duct giving way, the bile will have an exit. Occasionally a glass tube is used, when it is replaced the following day by a rubber tube, the latter being small enough to be carried down the glass tube and the glass tube withdrawn. When the gall-

bladder is so large that access to the cystic duct is difficult, it may be aspirated or opened and emptied, care being taken to guard against infecting the field of operation, when the removal, as above described, can be made. In some cases of gangrenous and phlegmonous gall-bladders the operation is made a little more difficult than in ordinary cases, yet this technic can be carried out.

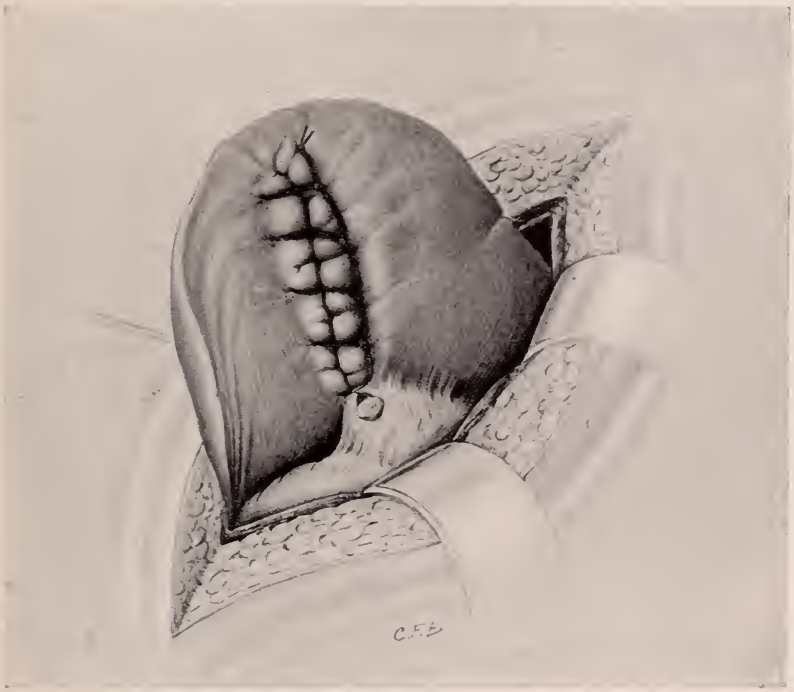

FIG. 10.—Gall-bladder bed sutured.

Drainage of the common duct by way of the stump of the cystic duct is only a temporary procedure. When, as is frequently the case, it is necessary to have prolonged drainage of the common duct, I open the common duct and introduce a T-shaped rubber drainage tube. Dr. Riesman and I have one patient who is wearing such a tube now going on three years. I have a number of

patients wearing these tubes. This form of drainage is introduced in certain cases of pancreatic lymphangitis and chronic interstitial and interacinar pancreatitis.

In passing, I beg to say that early drainage of the common duct by this method or by a cholecystoduodenostomy is the only chance for the cure of pancreatic diabetes. Metabolic studies will never cure pancreatic diabetes; only the early use of the aseptic scalpel and establishing drainage before the infection has caused a serious pancreatic lymphangitis, a forerunner of chronic pancreatitis, and this in turn of pancreatic diabetes.

DISCUSSION

Dr. A. P. C. Ashhurst: I think that any operative technic used by Dr. Deaver is worthy of consideration and discussion. I know how well he does his operations, and when you see them done by him they look as easy as the illustrations presented tonight. The trouble is that they are not so easy to the surgeon as the illustrations shown tonight would make them appear, and I should like to ask Dr. Deaver to tell us what he does in the more troublesome cases; what incision of the abdominal wall does he think gives the best exposure of the whole biliary tract? The bayonet incision of Kehr was used for years on the continent of Europe because it gave the best exposure to the ducts, and Terrier himself told me when I was in Paris some years ago that he had come to use that incision because he had tried the Mayo Robson incision and found it not sufficient. Most people in this country use the Mayo Robson incision, but in some cases it is not sufficient. The incision pictured in Dr. Deaver's illustrations appears to be an oblique subcostal incision. A second point is that if you find that the gall-bladder is very large you cannot get down to the cystic duct and the gastrohepatic omentum until you empty the gall-bladder. Dr. Deaver said nothing about emptying the gall-bladder first in these difficult cases.

One other point in technic is that if the patient is short and fat, the cystic duct when divided will retract at the distal end. I have had the experience of having the tube pull out of the common duct during the operation and have found it difficult to isolate the duct again and replace the tube. In the method described by Dr. Deaver there is no way by which one can hang on to the end of the cystic duct after dividing it.

Of course Dr. Deaver is describing a *typical* operation, and *typical* operations do not take account of the exceptional, the *atypical* cases. My only criticism would be that in the surgery of the biliary tract the difficult cases are so very frequent that they merit recognition.

Dr. Deaver, closing: Answering Dr. Ashhurst, I admit that some cases are more difficult to handle than others, yet I am always able to carry out this technic. I occasionally aspirate as well as freely open the gall-bladder to empty it. I find adhesions the most troublesome complication. The straight incision through the rectus muscle is the one that I most commonly use, yet now and then I make the Mayo Robson incision.

OVERACTIVITY AS A POTENT FACTOR IN THE ETIOLOGY OF SOME FUNCTIONAL AND ORGANIC NERVOUS DISEASES[1]

A STUDY OF ONE HUNDRED AND EIGHTY-EIGHT CASES

By ALFRED GORDON, M.D.

Several years ago, having had an opportunity to observe in my clinical work a large series of individuals working in lead, a problem presented itself to me for solution. Some workers showed left-handed wrist-drop, others right-handed; another had a paretic condition of the long flexor of the right thumb; another had a double wrist-drop; another had paresis of the right leg. In all these cases there was an absolutely identical etiological factor and still the functional disability did not manifest itself in the same portions of the body. In several professional pianists I have observed either attacks of cramp-like sensations in the wrist and in others in some of the fingers. The occupation and the continuous efforts being the same, the seats of the disorder are not identical.

In a large group of tabetic cases I had three cases of the cervical type in which ataxia began in the upper extremities. Why, therefore, is the functional disorder more marked in the arms than in the lower when the specific infection is of a general character? In some of my tabetic patients the eye symptoms were very conspicuous from the very beginning. In others the leg symptoms were the first to appear, and the eye disturbances appeared quite late

[1] Read February 2, 1916.

in the course of their tabes. In some of my cases the involvement of the sphincters was a late manifestation, in others a very early one. These differences in the onset and localization of functional disability in various individuals suffering from tabes dorsalis are striking and require an etiological explanation, especially in view of the same syphilitic toxic agent being present in all cases of tabes.

Why is it that paresis is most frequent in persons who exercise their intellectual functions more than their physical functions? I had four cases of juvenile paresis in whom the symptoms made their appearance with their entrance into college, otherwise speaking, at the time of greater demands on intellectual efforts. What explanation can one find with regard to such an onset?

In a group of cases of progressive muscular atrophy of spinal origin the affection began in the upper extremities in some patients, in the lower in others; in one limb in some cases and simultaneously in two symmetrical limbs in others.

The largest majority of my poliomyelitic cases were children in whom the affection was confined to the lower part of the body. In five adults the functional disability was observed in the one or both upper extremities. Three of these individuals were tailors and two were bricklayers. What is the reason of the difference in the localization of the pathological process in the children and adults?

In three cases of pernicious anemia in men there were evidences of a degenerative state of the spinal cord. In one case of the same affection in a middle-aged woman the central nervous system was not in the least involved. If a toxin is supposed to be the cause of the pathological process of the cord, wherein lies the difference in the cases of the men and of the woman?

These few examples of my entire series, the details of which will be given later, are sufficient to demonstrate the *raison d'être* of my inquiry. Weigert and Roux have long ago expressed the view that normally there is a state of equilibrium among all cells of the body, and their interrelation is such that when one cell becomes diseased or otherwise disturbed, the cells which lie close

to it develop an increased energy, namely, proliferation and, *per se*, repress the affected cell still further. Weigert also calls attention to the fact that the process of functioning of a tissue is accompanied by its destruction, which in normal conditions is rapidly compensated by a proper supply of nutrition. But if the latter is not forthcoming, the above-mentioned equilibrium is disturbed and degeneration takes place. Edinger (*Volkmann's Sammlung klinischer Vorträge*, 1894, No. 106), went further and said that if instead of or with a defective nutritive supply there is an excessive functioning of a cell, the increased growth of the neighboring cells will lead to a degeneration of that overactive cell which naturally is then less resistant. This is particularly seen when both elements are at work, namely, overactivity and nutritional deficiency.

Edinger succeeded in demonstrating the rationality of his contention by some experiments. By administering pyrodin to rats he produced in them a marked anemic condition resembling pernicious anemia of human beings. When this was accomplished by certain contrivances, he made them perform a considerable muscular activity. The result was that all anemic rats showed at autopsy a degenerative state of the spinal cord. The control animals, which after the injections of pyrodin remained at rest, presented a total integrity of their spinal cords. It is therefore evident that overactivity or superfunction is a powerful predisposing factor in producing diseased conditions in an individual whose organism is potentially under the influence of some toxic or otherwise abnormal element. Since function is apparently a potent agent in creating a syndrome of abnormal phenomena, it will be of interest to analyze closely a few of the disease processes of the nervous system from that particular standpoint.

In tabes, which is a syphilitic disease of the spinal cord, we have the organism of the indivudual possessing syphilitic toxins and consequently a disturbed metabolism. The great multiplicity of our sensory nerves which are peripherally situated, and thus receive incessantly impressions and transmit stimuli, are constantly at work. They play a very important role in muscular

contractions and in the awareness of position of muscles, of articulations, and limbs. The overactivity of these peripheral sensory fibers and of their prolongations—namely, posterior roots and posterior columns in a subject whose nutrition is deficient because of a metabolism which is disturbed through the presence of a syphilitic poison—is bound to result in degeneration of the overfunctionating sensory tracts. Hence the very frequent if not constant presence of symptoms depending on the function of the sensory tracts, namely, loss of regulating power of muscular contractions, of display of joints, loss of reflexes, anesthesiæ. The constant involvement of the posterior roots and columns (sensory) finds its explanation in the functional overactivity of those neurons. Moreover, the loss of coördination in the legs is observed in individuals who use and overuse the legs; loss of coördination in the arms is met with precisely in cases of arm or cervical type of tabes. It is also commonly known that in individuals who lead a sedentary life and overuse their eyes, the leg symptoms are not pronounced, but the eye symptoms are conspicuous. On the other hand, when a tabetic becomes blind, he commences to lead a sedentary life, uses his legs very little, and then we observe an improvement in the condition of the legs. When we turn our attention to the great frequency with which Argyll-Robertson pupil is met with in tabes, and take into consideration the great and constant activity of the light reflex in all human beings, it is easy to conceive its exhaustion and its disappearance when the organism is under the influence of toxins such as syphilitic in tabes. The same remarks may be made with regard to the muscles supplied by the third cranial nerve. Ptosis, for example, is one of the most frequent symptoms of tabes. It is due to a paralysis of the levator palpebræ superioris which is supplied by the oculomotor nerve. Its function is to keep the eye open, and it is therefore a very active muscle.

The various manifestations of tabes have been presented here for a discussion as to their mode of development. It appears that the conception of "overactivity" or "exhaustion" gives an adequate explanation for their occurrence. But it must always be

borne in mind that the "exhaustion" can be considered here only as a localizer, so to speak, of the morbid effect provoked by the presence in the organism of a toxin, syphilitic in the case of tabes, or else by the presence of other elements which are capable to place the tissues of the body in a pathological state. Let us now take up in detail the various affections of my series considered from the standpoint of exhaustion.

Occupation Neuroses. There were 37 patients distributed as follows: 12 stenographers, 5 newspaper reporters, 3 violinists, 6 pianists, and 11 shoemakers. All these patients without exception, presented the characteristic neurasthenic manifestations. For many months without interruption they strenuously carried on their work. Moreover, they all appeared to be underfed. In the stenographers and reporters the localization and character of the disorder were not identical. Some of them had the paralytic form, namely, a sudden sensation of fatigue and numbness in the hand while writing. In others there was an actual cramp, namely, a sudden extension of the index and flexion of the thumb. In others a cramp appeared in the wrist, but not in the fingers. One of the 3 violinists developed a cramp in the right hand and the other 2 in the left hand. Of the 6 pianists, in 2 a sudden paretic condition would set in the left wrist, the other 6 had a similar condition in the fingers of the right hand. Of the 11 shoemakers, 6 presented attacks of cramps in the biceps muscles of one or the other arm; the remaining 5 individuals had attacks of numbness in the thenar muscles of one or the other hand.

A close analysis of these 37 cases convinced me that the occurrence of the neurosis in one particular segment or portion of a limb was due to a predominance of effort practised by the affected region. Thus the violinists who had attacks of cramps in the left hand happened to be left-handed. The two pianists with the paretic attacks in the wrists were trained by teachers whose method consisted of holding the wrist stiff during the exercises.

The next group of cases in my series comprises 16 cases of *chronic lead intoxication*. Five of them had double wrist-drop. Two men had wrist-drop only on the right side, three men had wrist-drop

on the left side. Two men, typesetters in a printing house, presented a paretic condition of the three first fingers on the right side. Three men were house painters who used a small-size brush for their work. They presented wrist-drop on the right side. One individual was a supervisor in a white lead factory for ten years. He had to spend the entire day in the lead atmosphere. He developed a paresis of the right leg with a markedly diminished knee-jerk and tendo-achilles reflex. All the patients of this group had general evidences of plumbism, namely, blue line, obstinate constipation, albuminuria, headache. An inquiry into the character of the work of the 16 patients revealed the following: the individuals with double wrist-drop worked for a number of years in white lead factories where both arms were used with equal effort. The men with unilateral wrist-drop worked in lead colors, and their work consisted of mixing the dyes which they did with one or the other arm. Unilateral extensor palsies in plumbism is not a frequent occurrence. Nevertheless, in the few cases which I studied from the standpoint of exhaustion I found that the nature of employment coincided with the unilaterality of paralysis. The two typesetters, both right-handed men, used in their work chiefly the thumb and the first two fingers continuously. Although the general symptoms of saturnism were present, nevertheless the local symptoms were conspicuous in those fingers which are most persistently in use and therefore most readily fatigued. The three house painters being compelled to extend their wrist thousands of times in succession, naturally developed a paralysis of the extensor muscles of the wrist. All being right-handed, presented a right-sided wrist-drop. The supervisor of the lead factory who developed a paresis of the right leg had for duty to watch over groups of men. In making his rounds he would stop at a certain group and remain in one sitting position for an hour at the time. Close inquiry revealed that while sitting he would place the right ankle on the left thigh and keep up that position during the entire hour. He repeated this position at each group of men whom he was obliged to inspect. This attitude was considered as his habit, and he was never seen at the factory in another position. His wife

corroborated his statement, and said that this was also his sitting position in his own home.

The poliomyelitic cases comprise a group of thirty, among whom there were five adults and the others children. Of the former three were tailors and two bricklayers. One of the tailors had the right upper and lower limbs involved. Another tailor had both upper extremities at first stricken, but later the left arm recovered. The third had the right arm and right side of the face paralyzed. Evidently here the cervical cord, as well as the medulla, was affected. The two bricklayers had both their right arm and both lower extremities involved. The identity of the condition in both patients was striking. It is evident that the five adults whose chief exertion is manifest in the upper limbs had the cervical portion of the cord involved, as the ganglionic cells of this part of the central nervous system supplied nerve fibers to the brachial plexus, hence to the upper extremities. Out of the twenty-five children, ten had in addition to one or both limbs also one or both arms involved. A close inquiry revealed the fact that they belonged to very poor families and being of age from eight to twelve, they had to do considerable amount of manual work at home, such as scrubbing, cleaning, etc. Thus the involvement of the upper limbs can be readily conceived. On another page the usual occurrence of the poliomyelitic process in the lower extremities in children was mentioned. The reason of it lies in the great activity of the limbs, which are in a state of development in young children.

In a group of fifteen cases of progressive muscular atrophy of spinal origin the affection began in the lower extremities in one policeman and in one watchman; in two Italian street-cleaners in one upper limb. The remaining eleven cases showed an onset in both hands. The latter were all either laborers or tradesmen. The occurrence of the disorder in the lower limbs of the policeman and watchman speaks strongly in favor of the view in my thesis.

There were four cases of pernicious anemia in my series. In the men, three in number, evidences of degeneration of the spinal

cord were present. They presented symptoms of atoxic paraplegia with diminished or absent knee-jerks. Two of them were laborers and one a collector for an instalment house. The fourth patient, a woman, housewife, presented no disturbances of locomotion, and the knee-jerks were normal. Here again we have a corresponding link between the degree of overactivity of certain portions of the body and an organic lesion of that part of the nervous system which controls the affected portions.

Out of thirty-nine paretics, twenty belonged to the class of individuals who lead a purely intellectual life. Teachers presented the majority. In them the mental symptoms were the most conspicuous. With the exception of the eyes other physical symp- were very slight, namely, tremor of hands, slightly increased knee-jerks, and no toe phenomenon. Nineteen patients were tradesmen in whom the physical as well as mental symptoms were equally developed. They presented a condition called "taboparesis," namely, symptoms of tabes together with mental disturbances found in general paralysis of the insane. If we consider the combination of physical and mental efforts displayed in the occupation of tradesmen, we must admit that the above observation as to the morbid manifestations in these individuals is of no little interest and is more than a mere coincidence. Four patients of the entire group were cases of juvenile paresis. Their ages ranged from sixteen to twenty. The mental manifestations made their appearance at the time of their entrance in college. It is a period of intellectual life when application and mental efforts are tested at their utmost. It is also highly interesting to notice the variability of the eye condition in the paretics. Among the tradesmen there were two jewelers and three bookkeepers. It is remarkable that in everyone of these men optic atrophy and Argyll-Robertson pupils were very striking and developed early in the disease. In the persons who lead a purely intellectual life there were twenty-two teachers. All presented pupillary symptoms and some also palsies of the third nerve. Five cashiers of banks showed besides third nerve palsies, also evidences of eye-ground changes. The inference is consequently the same as in

the cases of tradesmen from the standpoint of the principle of "exhaustion."

In considering the group of cases of tabes, forty-seven in number, the inquiry was made as to the character of occupation and the degree of exertion of the tabetics. The largest majority (thirty-nine) were individuals who did a great deal of standing or walking and carrying heavy weights. In this group there were letter carriers, policemen, messengers, icemen, laborers, and motormen of street cars. The lower extremities alone were at first involved and the tabetic process began with disturbances of locomotion. Out of the remaining eight patients three presented at first ataxia of the upper extremities and diminution of knee-jerks, so that the diagnosis of cerebellar disease was originally entertained. Two of these patients were stenographers, and the third, a woman, worked at a laundry. The stenographers' duties were particularly laborious and their hours were very long. They eventually developed leg symptoms.

In connection with the subject of tabetic ataxia in the extremities it is well to recall what was said on the introductory pages with regard to the sensory neurons. The continuous and uninterrupted flow of impressions received by our peripheral sensory apparatus is the most important factor in all muscular contractions and in the display of our joints. Should the organism be invaded by a poison, such as syphilitic in tabes, the overfunctionating tissues or organs naturally will suffer first. Hence the disorder of locomotion and coördination which so much depend on the function of the sensory neurons. Proceeding further with the analysis of tabetic manifestations with reference to the principle of "exhaustion," the following is observed. Three patients who led a sedentary life worked in poorly lighted rooms, two of them, clerks in the same establishment, were assigned offices with windows opening in a very narrow and small yard, so that often they had to use gas-light. The third had to use gas-light all day; he was cashier in a small store, and his desk was in the back part of the store. All three when first seen presented optic atrophy without ataxia but with much diminished knee-jerks. Two patients were

electricians working in very brightly illuminated rooms and glaring light for ten hours daily. They also presented optic atrophy as an early tabetic manifestation. At the International Congress of Medicine held in 1913, the effect of very bright light was considered as injurious to the eye, owing very probably to the presence of ultraviolet rays. The early occurrence of optic atrophy in the two electricians is undoubtedly due to this latter circumstance.

Thirty-two patients of my entire tabetic series presented ptosis in one or in both eyes and Argyll-Robertson pupil in one or both eyes. If we consider the extraordinary activity and the incessant display of the light reflex from the moment we awake in the morning until we close our eyes for sleep—if moreover we consider the activity of the levator palpebræ which keeps the eye open—we cannot be surprised at the fatigue of these overactive functions in a body which is under the chronic influence of syphilitic toxemia; the early and frequent occurrence of Argyll-Robertson pupil and ptosis in cases of tabes are to be expected.

The present study, while it does not explain every morbid manifestation, nevertheless it is sufficiently suggestive as to the effect of organs on their early morbidity. It teaches that a body which is potentially diseased or which is under the influence of a disordered metabolism through the presence of a toxic material—will develop morbid phenomena primarily in parts functionally exhausted. The "exhaustion view," as we have seen, demonstrates quite satisfactorily that in syphilitic affections of the nervous system, in alcoholism, in plumbism, in acute infectious diseases such as anterior poliomyelitis, in profound anemias, and in other conditions accompanied by profound metabolic disturbances of any nature; it explains therefore the constant and almost uniform and earliest involvement of those portions of the body that are kept in a state of fatigue. This observation points to a very important feature in the domain of prophylaxy. Bearing in mind the pathological potentiality in cases of chronic toxemia, early treatment of the latter combined with removal from the usual occupation at the earliest possible moment is apt to prevent localized morbid manifestations such as we have seen above with regard

to locomotion and eye manifestations in tabes, or localized palsies in intoxications for example. A potential syphilitic individual will thus be able to avert for a long period of time the early oncoming of morbid manifestations of syphilitic or parasyphilitic character in the domain of the nervous system. The principle of prevention is here strikingly evident. The recognition and adoption of the view of "exhaustion" in affections of functional and organic nervous diseases appears to be far-reaching.

ON THE USELESSNESS OF SOME WIDELY EMPLOYED DRUGS[1]

By H. C. WOOD, Jr., M.D.

If there is any one fact which is clearly taught by the history of medicine it is the unreliability of empiricism as a test of therapeutic value. It is not necessary to recall to your minds the long array of various therapeutic fads and fancies supported by case reports, statistics, and all other kinds of clinical data, which have been eventually abandoned as worthless, and forgotten. It is true that there are a few drugs of recognized usefulness, such as mercury and quinine, whose efficacy was established long before there was any scientific explanation of their mode of action. But in these exceptional instances the enormous mass of clinical data running up into the millions of cases has been sufficient to outweigh the deceptiveness of experience. On the other hand, I hold it foolish to imagine that because some laboratory investigator has found that a certain drug does this or that to a dog or rabbit we should accept at once his deductions as to its clinical value. The proper standard of judgment as to the usefulness of any therapeutic agent should be a combination of experience and reason. Before we accept the value of a remedy there must be (1) a reasonable explanation of its mode of action, and (2) a reasonable amount of bedside testimony as to its usefulness.

I have for some time held the opinion that many of our common therapeutic practices are continued through habit or superstition rather than because of any benefit to the patient. In looking up the evidence as to the value of these agents I have been surprised

[1] Read February 2, 1916.

by its flimsiness and scantiness. I thought it might be interesting to give you some of the salient points of this investigation.

The first agent I want to call to your attention is the "Compound Syrup of the Hypophosphites," so widely employed as a general tonic, especially in tubercular conditions. The quantities of iron, of quinin, and of strychnin present in this preparation are so infintesimal that no one save the most fanatic follower of Hahnemann could attribute any virtue to these ingredients. The introduction of the hypophosphites we owe to Dr. Churchill, of Paris. About the middle of the last century Dr. Churchill conceived the theory that consumption was due to diminished oxidation in the body, and that the hypophosphites, being incompletely oxidized derivatives of phosphorus, would have a strong affinity for oxygen, and thereby increase the bodily oxidation processes. The objections to this theory are (1) there is no reason to believe that there is diminished oxidation in tuberculosis; (2) as a matter of fact the hypophosphites do not attract oxygen into the body even in sufficient quantities to oxidize themselves; and (3) even if Dr. Churchill's premises had been correct it would in no way argue in favor of the "Compound Syrup of the Hypophosphites" as a practical remedy. The amount of oxygen required to saturate the hypophosphites is so small that to cause an increase of 10 per cent. in the daily consumption of oxygen in an ordinary man would require a dose of about 4000 c.c., or about 1 quart four times a day. Still, it is not impossible that a man working along an erroneous theory might accidentally stumble on a real truth. The clinical proof that Dr. Churchill presented of the usefulness of this remedy was the *results in 35 cases* of tuberculosis, of whom 9 were classed by him as cured, 11 as improved, and 14 died. As for more recent clinical evidence, it is almost non-existent. In a brochure published by McArthur in 1881, and as he is commercially interested in this remedy he is not likely to have overlooked any favorable reports, the statement is made that the favorable conclusions are based upon reports of 259 cases by 30 different physicians. One can hardly regard this as an overwhelming mass of evidence after twenty-three years of trial.

Moreover, there is most convincing evidence that the hypophosphites do not have any effect whatsoever upon the system. In a bulletin recently published by the Ohio Agricultural Experiment Station, the result of an exhaustive study of the literature as well as elaborate experimental investigation, the conclusion was reached that of all the forms of phosphorus used in medicine the hypophosphites are the least readily assimilated by the body; as a source for bodily use they are inferior to sodium phosphate.

An unbiased study of the evidence, it seems to me, must inevitably lead to the conclusion that the compound syrup of hypophosphites has no more therapeutic value than a slightly bitter solution of sugar.

Some skeptical empiricist rises up to ask if this mixture is so impotent, why is it so widely employed? The reasons for its popularity are two. (1) The most important is the persistent advertising methods of the manufacturers of certain brands of the compound syrup of hypophosphites, and (2) its innocuous character. It is better to do nothing than to do the wrong thing, and when the patient insists on having some form of medicine, and the physician knows of no drug which is likely to be beneficial, he satisfies the longing of the sick man by ordering the compound syrup of the hypophosphites and salves his conscience with the thought that at least he has done no harm.

Opium as a Local Remedy. There is many a man who, while denying any accusation of being superstitious, nevertheless hesitates to make the thirteenth person at a table or has some pet which he regards as an omen of good luck. In a very similar way there are many physicians who profess to realize that opium has no local action but nevertheless persist in employing it as a topical remedy. And there may still be some superstitious enough to really believe that a drug so potent as a cerebral depressant must of necessity also have powerful local effects. Among the most common evidences of this belief may be mentioned that ancient, if not honorable, embrocation known as lead water and laudanum, the addition of opium to nutrient enemata to quiet the rectum, but above all the use of the opium suppository in various pelvic

inflammations. The first of these has perhaps a slight theoretic justification but the other two are as senseless as the incantations with which the ancients preceded their nauseating concoctions.

Crude opium has a marked local irritant effect. The *United States Dispensatory* says, "When long chewed it excites much irritation in the lips and tongue and may even blister the mouth of those unaccustomed to its use." Dr. Hill, in his *History of Materia Medica*, published in 1751, remarks of opium that "If kept long on the skin it takes off the hair, and it always occasions an itching in it; sometimes it exulcerates and raises little blisters if applied to a tender part," and recommended its use as a counter-irritant. I have myself seen two cases in which blistering followed the local application of lead water and laudanum, but whether as a result of this application I cannot say.

Of course a counter-irritant would likely be beneficial in various forms of arthritis in which Goulard's extract is commonly employed, and theoretically the counter-irritant effect of opium might be beneficial. To this sophistical defence, however, it may be replied in the first place we have a host of other counter-irritants more reliable and safer than opium; and, secondly, most of those who use it seem to do so with the idea that the opium will add some anodyne or antiphlogistic effect to the astringency of the subacetative lead.

The improvement which follows local applications of lead water and laudanum is due in part to the lint and bandages which hold it in place, perhaps slightly to the alcohol, but chiefly to the action of time which passes by while the application is left in place.

It may seem impiously iconoclastic to doubt the hallowed superstitions of the opium suppository. There appears to be in the mind of those who use it a subconscious delusion that as the rectum is nearer to the pelvic organs than the stomach, the opium must act more powerfully upon this portion of the body when given by suppository than when given as a pill. For opium placed in the rectum to reach the bladder it must be absorbed into the circulating blood and carried up to the heart and back again through the arterial system to the pelvis. Were it possible for the

morphin of an opium suppository to penetrate the layers of mucous membrane, connective tissue, and muscle which separate the interior of the rectum from that of the bladder, it would have no effect upon the latter organ, for the anodyne action of morphin, as of the other alkaloids of opium, is purely central. An opium suppository relieves the pain of a pelvic inflammation in the same way that a hypodermic injection of morphin does, by being carried to the brain and benumbing the perceptive centers.

Basham's Mixture. I have no statistics to determine the number of physicians who believe that "Basham's Mixture" is a sort of specific for Bright's disease. The fact that the popular names of the drug and of the disease begin with the same letter of the alphabet is to a certain type of mind evidence that they were meant to go together.

Some years ago at a time when it was believed that astringents might be absorbed into the blood the chloride of iron was recommended for the purpose of diminishing the quantity of albumin in cases of parenchymatous nephritis. According to present pharmacological and pathological theories, however, this seems neither possible nor desirable.

Of course I do not mean to deny the value of iron in some cases of diseases of the kidneys. There is no room for doubt that when nephritis is complicated with anemia, as it so often is, a chalybeate is beneficial. This, however, is beside the question; my contention is that iron in the form of "Basham's Mixture" or in any other combination can exercise no specific effect upon the kidney, but, as Tyson remarks in his *Practice of Medicine*. "It is prescribed constantly in the most reckless and thoughtless manner."

Dr. Basham, who originated the mixture of ammonium acetate with iron chloride, which goes by his name, was no believer in the antiquated astringent hypothesis nor yet in the specific antinephritic theory of the effects of iron, for he says in his work on *Renal Diseases*, published in 1870: "Preparations of iron are the best aid to the blood-forming function. But iron in any of these preparations cannot generate blood corpuscles. They can only be formed out of the nutritious elements of the food." In another place, "A long experience of these and other forms of renal disease

where the object of the treatment is similar has, however, convinced me that a soluble ammoniochloride obtained by acidulating liquor ammonii acetatis with dilute acetic acid and then adding the tincture of the perchloride is the most efficacious of all the so-called preparations of steel." I cannot assent to Dr. Basham's dictum that the solution of iron and ammonium acetate is the most efficacious preparation, for while there is no doubt as to its chalybeate power, its acidity and astringency make it peculiarly liable to disturb the digestive tract.

Lithia. The use of the salts of lithium in the treatment of gout was introduced by Carrod in 1861. He based his application of this remedy on the following hypotheses: The gouty paroxysm is due to the deposit of urates in the joint; the deposit is brought about by a diminished alkalinity of the blood which lessens the solubility of the salts of uric acid; and that lithia by a solvent action on the uric acid prevented the deposit. In support of the latter view he quotes the experiments of Uré who showed that 1 grain of lithia in an ounce of water would dissolve 2.3 grains of uric acid.

Neither his theory concerning the causation of the gouty attacks nor his explanation of how lithium would prevent them can be accepted. In the first place Magnus-Levy measured the alkalinity of the blood of twelve patients before, during, and after the gouty paroxysm, and failed to find any distinct change in its alkalinity. In the second place conditions accompanied with severe reduction of the body alkalinity, such as diabetic coma or leukemia, do not lead to a deposit of urates. In the third place, acidulating the blood cannot change the solubility of sodium urate without transforming it into uric acid, and the deposit in the joints is not of the acid but of the monosodium urate.

As regards the solvent properties of lithium toward uric acid, while it is true that uric acid dissolves more readily in concentrated solutions of the salts of lithium, nevertheless in the proportion in which it can occur in the blood it exercises no such solvent effect. Roberts has shown experimentally that the addition of lithium carbonate to the amount of 0.2 per cent. to blood serum had not the slightest effect in enhancing the solvent power of this medium

for sodium urate, and this amount of lithium would represent five times the fatal dose.

Some have attempted to attribute the supposititious beneficial action of lithium in gouty conditions to its diuretic powers or to its antacid action. As to the former, Good found that lithium chloride has no greater diuretic power than sodium chloride. As to the alkalizing properties, while it is true that by the administration of sufficient doses of the carbonate or citrate of this base, one can render the urine alkaline, the same thing is true of the corresponding salts of either sodium or potassium. Moreover, there is, to say the least, grave doubt as to the real benefit of alkalies in gout. Roberts says: "I have repeatedly administered the bicarbonate and citrate of potash continuously for three or four years in sufficient doses to maintain the urine persistently alkaline, yet I have seen the arthritic attacks recur with apparently unabated regularity."

It is to be noted that even if we accept Garrod's theories, the use of lithium must be limited to cases of typical gout with paroxysmal attacks of arthritis. Even the blind adherence to these improbable hypotheses affords no reason to believe in its usefulness in the various atypical manifestations of disturbed metabolism which we are in the habit of attributing somewhat loosely to the uric acid diathesis. By the strange irony of chance, in this country at least, the use of the salts of this metal is limited almost exclusively to this latter group of cases.

Aromatic Spirits. Some years ago the train on which I was traveling through New England struck a carriage containing an elderly couple. The man was not greatly injured, but the woman was pulseless and in a state of profound shock. They were both put in the baggage car and the train sped on its way toward the next town. In the interval I spent most of the time in giving hypodermic injections of digitalis and strychnin. When we arrived at the town the local doctor, who had been summoned by telegraph, was there to meet us. He stepped aboard the train, felt of the woman's pulse, opened his medicine case, poured out some yellowish liquid into a glass, gave it to the patient, who was by this time beginning to regain consciousness, and stepped back

saying with dramatic fervor, "Now you can move her with perfect safety." I was eager to learn what this wonderful remedy was which could so suddenly banish the evils of surgical shock, and so I looked over his shoulder as he replaced the bottle in his medicine case. It was labeled "Aromatic Spirits of Ammonia."

Having already committed so many heresies against the traditions of the elders, I am bold enough to sin once more and announce my disbelief in this time-honored stimulant. It is true that the salts of ammonia when injected intravenously in sufficient dose excite the vasomotor center and apparently also the heart, but there is no convincing evidence that they do so when given by the mouth. It is highly improbable that any salt capable of liberating ammonia ever finds its way into the circulation when given by the mouth. Weintraud administered as high as 9 gm. of ammonium carbonate by the mouth without any increase in the amount of ammonium nitrogen in the urine, but with an increase in the amount of urea proportional to the dose of ammonia. This change of ammonia to urea evidently takes place in the liver, for Starling has shown that the liver separated from the body has the power of converting ammonia to urea after injection of ammonia into the portal vein. It appears evident, therefore, that salts of ammonium taken into the mouth are carried directly to the liver and there rapidly destroyed before they can ever get into the general circulation. When given hypodermically, however, the conditions are somewhat different, for the drug must be taken up by the general circulation before it can be carried to the liver, and therefore must reach the vasomotor centers and the heart itself. Even under these conditions, however, its effects are so evanescent that it can be of practical value only in the most extreme emergencies. In the circulatory failure due to chloroform or in surgical shock one might conceive of a theoretic usefulness of the intravenous or hypodermic administration of ammonium carbonate or hydroxid, but when we realize that the effects of such an injection cannot last more than five to ten minutes, the folly of trying to maintain a flagging circulation in the later stages of pneumonia, for instance, by hypodermic injections of the drug every two or three hours is manifest.

HYSTERICAL MUTISM, WITH REPORTS OF CASES AND EXHIBITION OF A PATIENT[1]

By G. HUDSON MAKUEN, M.D.

Hysteria is essentially a disease of the mind. It is a psychical disease with many and varied psychophysical manifestations and symptoms. It has been variously defined as a "psychological disaggregation," a "psychological immobilization," a "psychological forgetfulness," a "psychological lesion" and a "disease of the personality."

There was a time when the diagnosis of hysteria carried with it a feeling of reproach, and the hysterical woman was regarded as one to be avoided and of being worthy of but little if any professional consideration. All this, however, has now changed and we have found that hysteria is an actual rather than a purely imaginary disease, that it is no respecter of sex, there being nearly as many hysterical men as women, and that many of both sexes afflicted are by no means lacking in efficiency and some of them occupy high positions of trust. Moreover, hysteria is a disease common to all races and to all ages, and even animals do not escape it.

The reversal of opinion with regard to the nature of hysteria is probably the result of a better understanding of the relationship which exists between the mind and the body, and there is probably no disease which so strongly emphasizes the closeness of this relationship.

The new psychology teaches us that psychical diseases are no less real and have causes no less definite than physical diseases,

[1] Read March 1, 1916.

hence the recent introduction of such terms as psychognosis and psychanalysis.

Freud's psychanalytic method, by which he attempts to establish a definite connection between such diseases as hysteria and their exciting causes, is now occupying the attention of the psychological and neurological world. It consists chiefly in an effort to ascertain the hidden causes of the various psychoneuroses by the use of so-called association tests and the analysis of dreams.

Hysteria has been frequently confounded with neurasthenia, and the difficulties of making a differential diagnosis between the two diseases are increased by the fact that they frequently occur simultaneously in the same individual. Hysteria differs from neurasthenia, however, in several important respects, one of which is the fact that the neurasthenic is introspective and deeply concerned about himself, whereas the hysterical individual usually appears to be quite indifferent as to his condition or its outcome.

Hysteria is said to be due primarily to a vulnerable suggestibility, and as most people are to some extent at least vulnerable in this respect, the affection may be latent in many hitherto unsuspected individuals. However, that may be, suggestion emanating either from within the individual's own consciousness or from some extraneous source, is undoubtedly a frequent exciting cause of hysteria. Traumatism also appears to induce hysteria, but as Doctor Dercum has pointed out, it is altogether probable that the causal factor in such instances is the emotional disturbance accompanying the injury rather than the injury itself.

Hysterical mutism is a form of hysteria which is by no means common. It bears a close resemblance to the various other manifestations of the disease, and occasionally it exists quite independently of all other hysterical symptoms.

Hysterical mutism differs from other forms of mutism in that it is of the profoundest character. As Charcot has observed, the hysterical mute is muter than the mute. The deaf-mute frequently makes some attempts at speaking, but the hysterical mute makes no attempt whatsoever. He seems like one who has never had the power of speech. He seems to have forgotten that he has any

organs of speech or that he ever had any. A similar psychical attitude characterizes those having other hysterical symptoms, such as hemiplegia, amaurosis, and deafness.

Intellectually, the hysterical mute appears to be somewhat below par. He appears to take but little interest in his own rather pitiable condition and to be able to make no effort whatsoever to overcome it. He has no cortical representations or memories of the movements required for speech production, and therefore he is like one who has never been in possession of the power of speech.

The psychological disaggregation or lesion is in relation with the motor rather than with the sensory mechanism. The patient usually understands well what is said to him, but, as I have said, he appears to have no power of oral expression and no psychical representations for it. He has no conceptions of the movements of the muscles employed in phonation and articulation.

In other words, the lesion, if I may so designate it, appears to be in Bastian's so-called glossokinesthetic area of the brain, where the memories of the muscular movements required for speech production are registered and stored. The kinesthetic memories, or memories for muscle movements, play an important part, not only in their relation to the speech processes, but also in relation to all the other motor processes of the human organism. But for such memories, walking, dancing, skating, and bicycle riding would all be impossible, and the acquirement of any kind of language by the deaf would also be impossible. It would appear, therefore, that too little attention has been given to this kinesthetic, or sixth sense, as it has been called, as a factor in psychophysical development.

Hysterical mutism differs from hysterical aphonia in that in the former condition the patient is both aphonic and inarticulate, while in the latter he is merely aphonic and can speak fairly well in whispered sounds. The hysterical mute is not only aphonic, and can speak fairly well in whispered sounds, but he cannot even whisper, a condition which Dr. Solis Cohen has designated as apsithyria. The hysterical mute, therefore,

is aphonic, apsithyric, and inarticulate, the mute condition differing from the aphonic and apsithyric in that it involves all the motor mechanisms of speech, including even the respiratory mechanism as it is used in voice and speech production.

As I pointed out in a previous article, the terms aphonia and mutism have been used in scientific discussions with too little regard for their exact meaning. Aphonia should be used to designate a condition of complete phonatory disability, while the term mutism should be reserved to designate a condition of complete phonatory and articulatory disability.

I have also called attention to the fact that aphemia or motor aphasia is often confounded with mutism, but the aphasic are not altogether mute. They are usually able to produce occasional sounds or words, and this may serve to distinguish the conditions. Moreover, mutism is sometimes the result of actual organic lesions of the brain, and thus it may be very like aphasia, and the differential diagnosis may be difficult. It should be borne in mind, also, that the two diseases may exist simultaneously in the same individual, thus complicating still further the question of a differential diagnosis.

As I have said, hysterical mutism is not in my experience a very common disease, although Marcel Natier, of Paris, had collected seventy-one cases more than twenty-five years ago, and the affection has been studied in England and on the Continent by some of the most careful investigators. It is said that the term hysterical mutism was first given to the condition in 1883 by Professor Revilliod, of Geneva, who wrote a paper on the subject which attracted much attention. Then followed papers and lectures on the subject by Charcot, Cartaz, and H. Bok, of Berlin.

I myself have seen only two well-defined cases, the first being one reported before the American Laryngological Association in 1906. The patient was referred to me by Dr. John K. Mitchell, and his condition was carefully studied by several other Philadelphia specialists, including Dr. Weir Mitchell. For my full report of the case, I refer you to the *International Clinics*, 1, 17th

series, and Doctor Mitchell's report of the same case may be found in the *Journal of Nervous and Mental Diseases.*

Briefly, this case was that of a man twenty-two years of age, who had received a severe head injury, rendering him partially unconscious for a few moments, after which he complained of headaches and right-sided tinnitus aurium with complete loss of speech. The family history was negative, and two years previously the patient had an attack of typhoid fever, which disease, it is said, sometimes induces hysteria in those predisposed to the affection, and he also had a severe attack of tonsillitis, which may have been an additional causal factor. The patient complained of sharp, shooting pains throughout the body, he had lost twenty-five pounds in weight, there was great mental depression, and his sleep was much disturbed by dreams. Sensations were normal externally, as were also the reflexes and electric reactions. There was no muscular incoördination except in the mechanisms of speech. The eye-ground was normal, except for a complete reversal of the red and green color fields, a condition which is supposed by many to belong to hysteria, although it is occasionally found in those having no other hysterical symptoms. There was a left-sided intranasal pressure, the removal of which seemed not to influence the condition. The mutism continued for several months, in spite of everything that we could do. All the known methods for relief were employed, including suggestion in both the waking and hypnotic state, until one night at the Orthopedic Hospital he had a kind of nightmare or hysterical fit, during which he uttered several sentences with normal phonatory and articulatory precision, and he continued to talk with the resident after he had recovered from the attack. The following morning his speech was normal, and it has remained so for the several years during which I had him under observation.

This was clearly a case of so-called traumatic hysteria, and the hysterical symptoms which resisted all our efforts yielded to the emotional excitement of an explosive nervous attack.

An interesting feature of this man's recovery was noted by Doctor Mitchell in his report of the case; together with the return

of the faculty of speech there was also found a complete absence of any change in the color fields. In other words, this particular symptom or sign of hysteria disappeared together with the mutism during the hysterical seizure.

My second case is the one I have exhibited this evening. The patient was referred to my clinic by Dr. Curtis C. Eves, and Doctor Rhein also examined her in the Neurological Department of the Polyclinic Hospital. I may say that we have all had difficulty in getting the facts in connection with the early history of the case, because the mother was ill at the time, and either does not have a clear understanding of it herself or she is incapable of giving a satisfactory explanation. The mother's own intellectual faculties are not of the highest order, and both she and the father of the child are of a nervous type. Their symptoms, however, are vague, and in answering questions relative to the child's early history they act as if language existed merely for the purpose of concealing thought. This child unquestionably inherits a neurotic temperament, if not a definite tendency toward hysteria, and as far as I have been able to ascertain, her home environment is of such a nature as to encourage and develop this tendency.

From a psychical standpoint, hysterical children are of two classes. One is characterized by those manifestations embodied in the word precociousness, while the other and a much smaller class evinces mental inaptitudes or backwardness. This child has always been somewhat dull mentally, but she is more so since her illness. She is thirteen years of age, and she has always been more or less subjectively minded and melancholy. About three years ago she had four convulsive seizures which were preceded for several months by failing health with some loss of power in her limbs, presumably as a result of a severe fright from a threatened railroad accident. The convulsions continued intermittently over a period of four hours and left her entirely helpless.

For about a year she was confined either to her bed or rolling chair, where she took her food and medicines only under compulsion, and she would not or could not talk at all. Her physician regarded her as hopelessly ill and said he could do no more for her.

Then the mother says that in desperation she got her out of bed and made her walk, from which time she gradually regained the use of her limbs, but the mutism continued, and I saw her first in January of last year.

I found her to be a rather meagerly developed child, both physically and mentally. She was nervous and excitable, but extremely reticent. She would make but little effort to answer questions, but she could write short words fairly well. She had a tendency, however, to transpose certain of the letters of the words. For "man" she wrote "nam" and for "Ida," her first name, "Iad."

Psychically, she seemed somewhat obtuse, although her disinclination or inability to answer questions upon examination makes the history of her mental condition unsatisfactory. A significant fact, however, is that whereas prior to her convulsive seizures in 1912, she had reached the fourth grade in school, she is now back in the second grade.

Neurologically, it was found that many of the usual phenomena of hysteria were absent. Aside from her speech mechanisms, she had no motor paralyses or incoördinations, and she had no muscular contractions or peripheral disturbances of sensation. On the other hand, the knee-jerks were increased on both sides, but curiously enough there was on the right side a positive Babinski, which points to a previous organic lesion rather than to hysteria, and as Doctor Rhein has suggested, this Babinski reflex throws some doubt upon the hitherto supposed hysterical character of the convulsions and former paralyses. The mutism, however, was complete, and as Doctor Lloyd has well said, it must be hysterical because it cannot be anything else.

Ophthalmologically, it was found that the eyes were normal interocularly and extra-ocularly, except for a high refractive error, but there was a contraction of the form fields and, as in my other case, a reversal of the color fields, the latter two conditions being somewhat characteristic of hysteria.

Laryngologically, I may say that there was no loss of motion in the cords, but there was no adequate control of them for purposes of phonation. They would come into apposition immediately,

to separate again when any attempt was made to use them in a purposeful way.

One of the tonsils had been removed, but the other one still remained unobstructive and deeply imbedded. In other respects the nose and throat conditions were normal. The interrupted current had been applied on several occasions with negative results, and when she came to me she appeared to be convinced that she could not speak and that it was utterly useless to try.

The treatment in this case consisted first in an effort, as someone has said, to "scare back" the voice and thus to get even a slight phonatory sound upon which to build still further, but nothing seemed to scare or startle the patient, and when very strenuous efforts were made she resorted to tears. There was no phonation, however, in her crying or laughing or even in her coughing, and we were unable to elicit a single sound.

Then we tried to develop whispered speech, and after several weeks of training she was able to make herself understood in this way and to engage in conversation with her friends in a satisfactory manner, so that she and her mother were delighted with her progress.

It was apparent to us, however, that further attempts to help her to bring back her voice were futile so long as she remained in her home environment, and we were about to arrange to place her in the hospital for a course of treatment when some of her friends took her to a religious camp meeting, where she was induced to "pray back" her voice. The enthusiasm and excitement, I am told, were intense, and she was urged to pray long and loud. This happened to be the thing that seemed to be necessary to bring about a mobilization or aggregation of her psychical forces and enable her to speak with a laryngeal sound after many months of complete aphonia. It may be contended and probably is so contended, by those who practise this form of the healing art, that the cure was purely in answer to prayer, and we must at least concede, I think, that the exercise of faith on the part of the child was probably an important factor in this psychic change.

Unfortunately, however, her conversion has not cured her of all

her ills, because even now she is at times extremely irritable and depressed and says that she is sorry that she did not die two years ago when she was so very ill.

Manifestly, the hysterical tendency or temperament still continues, and fresh outbursts of hysterical phenomena may be expected unless a complete change is made in her environment and manner of living, and unless some form of psychophysical training is adopted for the purpose of enabling her to get a better control of herself.

She is still following up the religious precepts which she learned at the camp meeting, and she has persisted ever since in conducting morning and evening prayers with her family. Only the other day she gave us an example of her devotional propensities by kneeling with me and my assistants in my office and praying for us and things in general. Her petitions while not clothed in the best of language, were nevertheless fervent and to the point.

Since writing this report several weeks ago, and after having gone over the case neurologically with Doctor Rhein, who, by the way, discovered for me the Babinski reflex, which is so characteristic of an organic lesion of the central nervous system, I renewed my efforts to get a fuller history of the child's earlier illness. Incidentally, I found that she had been a patient at the Orthopedic Hospital, and upon application at this institution I learned that she had previously been taken to the out-patient department of the Episcopal Hospital, where a Wassermann reaction was found to be positive. Whereupon another Wassermann test was immediately made for me in the laboratory of my friend, Doctor Ludlum, and it also proved to be positive.

These pathological findings obviously suggest very interesting complications, and they naturally lead us to change our diagnosis so far as the original convulsive seizures and hemiplegia are concerned, but they can scarcely explain the mutism with its peculiar history and manner of cure.

The synchronous existence of functional nervous diseases with organic diseases of the brain is by no means uncommon, and as Doctor Burr has pointed out in a recent article on the subject,

their concurrence in some instances may be purely accidental, while in others "either the organic disease is itself the exciting cause of hysterical symptoms in a congenitally predisposed person, or both the organic and the so-called functional disorders arise from the same exciting cause." All this, however, is more or less speculative, I suppose, and we only know that in this particular instance, as well as in many others, the two forms of disease (functional and organic) do exist in one and the same individual.

That the mutism was hysterical I think there can be no doubt, and it may be well among the possibilities to suppose that the syphilis, whether congenital or acquired, may have been a causal factor in the development of this hysterical symptom in much the same way that any emotional shock, for example that resulting from trauma, may give rise to hysterical manifestations. In closing, I shall be content to give a brief summary, together with my conclusions.

Summary and Conclusions. Hysterical mutism is a somewhat rare affection, but it occurs at all ages, in all races, in both sexes, and even in animals.

Hysterical mutism is usually of sudden origin and the result of some severe psychical or emotional shock.

Hysterical mutism is characterized by the complete absence of the psychical representations necessary for the production of speech.

Heredity is an etiological factor so far as it furnishes the neuropathic or psychopathic soil for the development of the affection.

The mutism generally occurs in conjunction with other hysterical phenomena, although it may be the only hysterical symptom.

Hysterical mutism may occur in conjunction with organic nervous diseases which themselves simulate hysteria, and it can be distinguished from them only by actual discovery of the pathological conditions giving rise to the particular affection.

Hysterical mutism differs from hysterical aphonia in that the former evinces a more extended disability of the mechanisms of speech, and it differs from aphemia or motor aphasia in that the mutism is absolute.

The treatment of hysterical mutism may be either brusque in character or gentle and persuasive. An effort should always be made to "scare back" the voice, and then by psychophysical training to reëstablish correct methods of speech, but failing in this somewhat abrupt and severe treatment, milder educational measures should be used, such as are embodied in the terms suggestion, persuasion, and reëducation.

I myself can scarcely be regarded as one capable of speaking authoritatively upon the treatment of hysterical mutism inasmuch as neither of my two cases was entirely cured until extraneous influences were brought to bear upon them, one patient regaining his voice during an hysterical nervous attack, and the other during other extraordinary emotional excitement.

In both cases, however, the final successful issue was probably made possible by the psychophysical training which led up to it, and I am convinced that my failure to actually cure my patients was due to my lack of ability to arouse in them the psychic representations necessary for the production and externalization of the symbols of speech.

I may say that if physicians generally were better practised in the art of psychotherapy, there would be fewer so-called miraculous cures reported outside the pale of the regular profession, and it is probable also that a wider and more accurate knowledge of hysteria as a disease would enable physicians to have a better understanding of the phenomena which appear in the more serious psychoses.

DISCUSSION

Dr. James Hendrie Lloyd: Dr. Makuen's case brings out very clearly some of the problems involved in certain disorders of speech. It is necessary to distinguish *mutism* from *aphonia*, and *aphasia*, and finally *dysarthria*. Mutism is the entire suspension of speech, not its abolition. The patient has not lost the power to speak, but simply the will to speak. There is no paralysis of the organs of speech, but speech is simply in abeyance. The patient makes no attempt to speak, but simply remains

dumb. The affection, in other words, is entirely mental. As a rule no persuasion, cajoling, or threatening has any effect to cause the patient to use the vocal organs. There is no response on the part of the patient, no effort, no whispering, no movements of the lips, no pantomime of speech.

Aphonia, on the other hand, is not the abolition of speech, but the abolition of voice. The two things are very different. The aphonic patient does not vocalize. The affection is local, that is to say, its manifestation is local, or entirely in the larynx. The larynx does not functionate or produce voice, but the supplementary organs of speech, such as the tongue and lips, may functionate, hence in many of these patients attempts to talk are made, and the lips may move as in talking, but no voice is produced. The patient sometimes may whisper, and very often goes through the pantomime of talking, without uttering a sound. She even seems sometimes to want to talk, but cannot control her larynx to vocalize.

Motor aphasia is due to an organic brain affection, in which the patient has lost the memory or the power of making speech. It is not due to a paralysis of the organs of speech. The patient can still vocalize and may even be able to utter a few words, but his cortical speech centers are affected, and he cannot make words in his mind. There are many varieties, but it is not necessary here to define them.

Dysarthria, also due to a brain lesion, is caused by a paralysis of the muscles of speech, the lips, the tongue, and the vocal cords. It is probably best seen in pseudobulbar palsy. The patient cannot talk because his speech organs are paralyzed. It is not an aphasia.

These various disorders of speech are the symptoms or effects of entirely different affections. The mute patient is one thing, the aphonic patient another thing, and the aphasic patient still another. It is mighty important to recognize these differences.

Mutism is the symptom, as a rule, of a profound mental disorder. It is most commonly seen in the affections known as stuporous melancholia, or melancholia attonita. It may also be seen in the catatonic form of dementia precox. Cases have been recorded in which it had lasted for years, to end in recovery. A few days ago I saw a woman who was beginning with her second attack of atonic melancholia with mutism. No persuasion availed to make her speak. Seven years ago I saw her at her home, in Stroudsburg, beginning with her first attack. She was brought to the Pennsylvania Hospital for the Insane, where she remained mute for nearly a year, and then recovered. The mental state in such a patient is hard to understand; it is one of profound depression and negativism, but as a rule there is no dementia, for a good recovery often follows a prolonged attack. Sometimes the recovery of speech is sudden.

In the catatonic form of dementia precox there may also be a state of negativism, with stupor, catalepsy, and even mutism. Dr. Makuen's case raises the interesting question whether his patient has really a form of dementia precox. In some of these precocious forms religious emotionalism may be seen, as in his case. Syphilis in the young may also possibly cause a brain infection which manifests itself very much like a case of dementia precox; in fact, one of the nice questions now before neuropathologists is the possible relationship of syphilis to dementia precox.

That mutism, such as I have defined it, may be a symptom also of hysteria seems to be established by the testimony of some competent observers. Dr. Makuen has referred to a number of these, some of them, indeed, very eminent names. Aphonia, however, is the form of speech defect most commonly seen in hysteria. Personally I am so much convinced of this, and of the comparative rarity of pure mutism in hysteria, that in any given case of alleged hysterical mutism I should want to be fully convinced that the more grave psychoses, such as stuporous melancholia and dementia precox, had been carefully excluded. Dr. Makuen has discussed some of these fine points in diagnosis, about which perhaps it does not do for any of us to be too dogmatic. His case illustrates how these questions may arise.

The organic speech defects, such as aphasia and dysarthria, form a class apart and cannot be discussed in brief space. It is usually not difficult, however, to differentiate them from the so-called functional disorders, such as mutism and aphonia.

Dr. Alfred Gordon: Apart from the mental affections, such as catatonic forms of dementia precox and stuporous insanity where the mutism can be easily detected, and where the mutism is one of the symptoms of the general picture of the disease, the condition presents no difficulty in recognition. We know that functional disturbance in the field of sensory disorders is not an infrequent phenomenon in connection with organic disease of the nervous system. An illustration of this was seen in one of my recent cases, that of a young woman of thirty-two whose Wassermann on the blood and spinal fluid was strongly positive. There were optic neuritis and palsy of the third nerve with total hemianesthesia on the left side, the scalp, face, ear, arm, and leg were totally anesthetic; there was also concentric contraction of the visual field on the left side. The question of organic disease arose in view of the Wassermann reaction, the optic neuritis and the difficulty of micturition, the character of the sensory disturbances and other symptoms. The loss of sensation and contraction of the visual field so characteristic of hysteria made me rather reject the possibility of organic origin of the hemianesthesia. The

patient was kept under further observation and one day the fire engine passed in the neighborhood. The woman was badly frightened and she became totally mute. When I observed this, my idea of the hemianesthesia being functional in nature became stronger in spite of the Wassermann and other symptoms of organic involvement of the nervous system. Two weeks later the woman had a quarrel with her mother-in-law, and immediately afterward she recovered her speech entirely. Here was a case of complete mutism accompanied by hemianesthesia, typically hysterical mutism; an emotional element entered and the mutism disappeared. In my judgment mutism occurring under like circumstances is in 99 per cent. hysterical. Charcot liked to demonstrate this condition by the case of a woman who had hysteria and who for many years suffered from astasia-abasia, although there was no distinct paralysis. With treatment she would improve and then relapse, becoming entirely impotent. One night he called in his internes and arranged that at two o'clock in the morning they should sound the fire bell. A few days later this was done; everyone knew it was the fire bell and the woman jumped out of bed and began to walk. She continued to improve but relapsed again. Here again was a case of a functional disorder with improvement upon the entrance of an emotional element. Such cases I believe are unquestionably hysterical. In Dr. Makuen's interesting case I believe that parallel with the organic condition, which was indicated by the Wassermann and the presence of the Babinski sign, there was a distinct typical hysterical mutism.

Dr. Charles K. Mills: The subject of the papers this evening is to me one of great interest. With regard to mutism, as defined by Dr. Lloyd and discussed here, when it is present in a case of mental disease it is due to a profound, delusional, depressive state, and we are usually able to recognize the existence of this delusional condition by concurrent symptoms. If hysterical aphonia in a broad sense may be regarded as delusional, it is nevertheless distinctly different from the mutism of well-defined mental diseases and needs to be studied independently of the latter. In this case the aphonia or mutism is due to suggestion—it may be sometimes to autosuggestion which, however, may have had its origin in outside suggestion. In the hysterical case the suggestion acts especially upon the cerebral volitional mechanism rather than upon that logical mechanism, the disorders of which produce delusions or obsessions at the basis of insanity proper. Treatment should be instituted with these views in mind. It is extremely difficult to say just how to treat the cases of mutism of a delusional origin. If not self-limited, they at least seem to be limited by some influences beyond our comprehension.

The cases of true hysterical aphonia are open to more definite treatment,

one based very largely upon the use of strong suggestion. This, however, may be accompanied by other methods. In my own practice I have frequently used a form of respiratory treatment, vocalizing or attempts at vocalizing associated with forced respiratory movements which sometimes serve to bring back the speech. This has sometimes proved very efficacious in connection with other methods of suggestion.

With regard to the cases of syphilitic hemiplegia referred to by Dr. Gordon, which is of interest in connection with this discussion, undoubtedly there is present not only a syphilemia, as in the case alluded to and probably in Dr. Makuen's case, but in addition a hysterical or functional affection. With this in mind the explanation is not so difficult. The influences of the toxic agents upon the central nervous system are such as to reduce the cerebral powers of resistance and render the brain of the individual more susceptible to impressions from without and suggestions from within. The treatment in such a case should be in the first place that which is directed to the relief of the blood state by the best possible modern methods, and in the second place, by the use of strong suggestion.

Dr. John H. W. Rhein: I examined the case described by Dr. Makuen at his request at the Polyclinic Hospital and found the same difficulties as Dr. Makuen in eliciting a history. At the time of my examination I failed to find any evidence of hysteria and the examination was negative except that the Babinski sign was positive on the left side, the side on which she had previously had hemiplegia. In view of the fact that the Wassermann reaction was positive I feel justified in saying that the child probably had an organic lesion at the time at which she suffered from hemiplegia. I am inclined to explain the symptoms referable to the mental condition and mutism as due to a specific psychosis plus syphilitic cerebral spinal involvement or possibly as Dr. Lloyd suggests a dementia precox. I do not believe the symptoms were entirely hysterical.

Dr. Makuen (closing): I had hoped to have a fuller discussion of the differential diagnosis between hysterical mutism and motor aphasia. As I have said in my paper, the only objective difference seems to be that in the former the mutism is complete, while in the latter the patient usually gives expression to more or less disconnected words and phrases. The subjective difference between the two conditions, of course, is clear enough, hysterical mutism being a purely functional disease while motor aphasia is usually of organic origin. Dr. Lloyd says that the patient has the power but not the will to speak, whereas it seems to me that even the power to speak is lost. In the 2 cases which I have studied very carefully, both desired very much to speak, and both tried very hard to

do it, but they seemed to have forgotten how, and it was as if they had never known how. They appeared as if they had never spoken, and therefore were unable to do it. They both finally learned the articulatory movements necessary for the purpose of speech, but they were unable to produce any vocal sounds whatsoever, and therefore I concluded that they had lost the necessary psychic representations for phonation, and try as they would, they were unable to arouse them. It is true, as Dr. Rhein has said, that the long illness in the case of the young woman whom I presented to you may best be explained as being the result of the specific infection, as shown by the positive Wassermann, but as to the hysterical character of the mutism I think there can be no doubt, and the changes in the color fields, although not pathognomonic of hysteria, are at least suggestive of this condition. As to the mutism, however, it must be hysterical because, as Dr. Lloyd says, it can scarcely be anything else, and furthermore, as I have said in my paper, it is not unusual to have one or more hysterical symptoms appear in connection with organic disease.

SOME FACTS AND FALLACIES CONCERNING ABDOMINAL ADHESIONS AND BANDS[1]

By GEORGE G. ROSS, M.D.

AND

J. BERNHARD MENCKE, M.D.

With the continued development of abdominal surgery there has been an increasing appreciation of the importance of the normal activities and pathological conditions of the peritoneum. The recognition of gross lesions of this structure such as the more evident forms of active peritonitis and the resultant inflammatory adhesions antedates, by far, our knowledge of the condition giving rise to them. The demonstration of the bacterial origin of peritoneal lesions and subsequent investigations as to the causation of such bacterial invasions have placed our knowledge of the more acute forms of peritoneal disease upon a firm basis.

A far more difficult problem presents itself when we consider peritoneal adhesions and bands for which it is impossible to ascertain a definite antecedent acute inflammatory lesion. The recognition of such structures is not a recent one. It has been stated that Virchow, in 1858, was the first to describe these structures more in detail, but at that time, of course, there was no basis for a correct opinion concerning their causation. For a long time after the beginning of abdominal surgery upon a larger scale, the avoidance and treatment of acute peritonitis engrossed the attention of operators. Further and extended observation of early cases led to information concerning postoperative and post-

[1] Read March 1, 1916.

inflammatory adhesions and to efforts to determine their cause and to attempts at their prevention. A still later development of the matter was an endeavor to understand the symptomatology and diagnosis of abdominal adhesions as apart from other lesions.

Finally, the work of such men as Jonnescu, Lane and Treves, several decades ago, called the attention of surgeons to the existence of various folds, bands, and membranes in relation to portions of the gastro-intestinal tract and to their possible importance. It remained for J. N. Jackson, in 1909, to bring to a definite standing in our minds the significance of certain pericolic structures or membranes, and following him many investigators and clinicians have endeavored to place upon a firmer basis our knowledge of this subject.

A great deal of confusion which exists concerning all these adhesions, bands, membranes, etc., may be obviated by the clearer grasp of certain fundamentals and an effort to distinguish between various groups and varieties of these structures, instead of attempting to make one theory as to their causation explain every case.

All abdominal adhesions and bands may be grouped into two classes:

1. Those adhesions and similar structures whose character and location make it evident that they must have been the result of a definite attack or attacks of peritonitis of more or less severity.

2. Those structures in which some doubt may be entertained as to their formation as the result of a distinct peritonitis.

The former class gives us more data upon which to form a conclusion; the latter is the one about which the discussion among surgeons at present centers. Jackson himself has stated that he is in the dark as to the causation of the membrane named after him or Jonnescu; others have been more definite in statements as to their opinions.

Membranes and adhesions not frankly the results of a peritonitis may be again subdivided into

(*a*) Structures whose character and location make it possible that they may be the results of fetal maldevelopment, and

(*b*) Similar membranes in localities in which this causation is not so plausible.

The explanation of the existence of both of these types has been attempted upon numerous grounds. Briefly, they are:

1. The attempt to explain all or most of these formations on the basis of failure of the rotation or descent of the gut or an anomalous growth of the peritoneum and mesentery in the presence of normal rotation. While there may be some basis for this assumption in certain cases, it has appeared more than a coincidence to us that these bands or veils with their attendant symptoms should be so often localized at or near that portion of the gut where we are most likely to have lesions of an inflammatory nature. Nor is it a counterbalancing fact that there is not to be found the remaining proof of such an inflammation.

Eastman, in experiments on rabbits, by artificially producing coprostasis, has succeeded in producing a low-grade peritonitis and subsequent formation of membranes. Cheever is one of those who dissent most strongly from this view of the matter and his dissections somewhat strengthen his position. Nevertheless, the experiments mentioned and those of Murphy and others on intestinal obstruction lead us to the belief that in many cases a migration of bacteria through the intestinal wall can, and does, give rise to diffuse peritoneal inflammation of a low grade which results in the formation of veils, membranes and bands.

It is a remarkable fact that these structures are most often found in adult life and at that time give rise to symptoms. Is not this also an evidence that an early underlying cause gives rise to conditions which cause their symptoms later, when the membrane or other structure becomes of such extent as to seriously interfere with the activity of the gut?

Granting at once that certain of the structures might possibly be the results of causes unassociated with inflammatory changes, there is no evidence that this is the case and it is far more reasonable to suppose them the results of an inflammation until the reverse is proven.

2. Mr. Arbuthnot Lane and others (Fagge, etc.) have attempted to explain peritoneal bands as "crystallization of lines of force" and the result of stress upon normal structures, causing them to be altered. There is no doubt that malposition of abdominal viscera

must bring with it a malposition of their surrounding structures and coverings, but the whole of Mr. Lane's explanation of these conditions is surrounded by a haze of theory, argument, and supposition unsupported by facts. His theories are curious and interesting but entirely unconvincing; plausible on first reading but not susceptible of close reasoning. Some of his views and statements do not bear analysis at all; their true inward meaning must be known only to Mr. Lane himself. To explain a definite and often pathological structure upon such vague and fanciful grounds does not tend to clarify the situation; it only introduces an additional mystery to a difficult subject.

3. In contradistinction to these views there are those who would explain practically all of these new malformations of the peritoneum as the results of inflammation. It is a matter of no great consequence that many of these adhesions or bands are or seem to be congenital. The evidences of antenatal inflammation of the peritoneum are sufficiently definite in numbers of reported cases (Doran, Kieth, Ballantyne, and Veszprenit). It is the opinion of these men that such instances of fetal peritonitis as they cite are by no means uncommon; and the examples given are those where there was no evidence of a purely mechanical origin of the peritonitis such as a perforation. The transmission of toxins and drugs from mother to fetus is universally acknowledged, as is the transmission of the infecting agents themselves by means of the blood or indirectly through the fetal appendage (Burnet).

Since then the existence of not infrequent antenatal peritonitis is proven, we believe that those adhesions which are found not to have been caused by a peritoneal inflammation after birth are the result of a similar condition in the fetus; that whereas many of these adhesions are congenital they are not developmental in origin. It must be understood that from such an origin we exclude those instances in which there is but an accentuation or simple enlargement of a peritoneal membrane, fold or process normally present.

In taking inflammation, acute or subacute, as a causative factor of a condition we are dealing with a fundamental entity, a condition with the results of which we meet every day. In

assuming developmental errors as causative factors of lesions evidently pathological we can only adopt the absence of other demonstrable causes as proofs of a supposed cause.

A consideration of the opinions of various clinicians and experimenters makes evident the fact that there is no agreement concerning these bands or membranes.

Fallon even states that Jackson's membrane is always present and is a normal structure. With this view we are not at all in agreement. Lynch, after citing certain facts to prove that Jackson's membrane is not of inflammatory origin, concludes "that the membrane described by Jackson is of embryonal origin, but it may be so altered by inflammation as to appear like an ordinary adhesion." It may be embryonal and nevertheless inflammatory, and to distinguish between a membrane of inflammatory origin, antenatal or postnatal, and a membrane merely altered by an inflammatory process appears to us to be impossible.

We think that there is every reason to believe that practically all of these structures are the result of peritoneal inflammation of some kind. And moreover, we are convinced that more extended clinical observation on the part of those who have had greater opportunities to theorize about, than to observe, these formations can only serve to make them agree with this opinion.

Concerning adhesions which are frankly the result of one or more attacks of peritonitis after birth a great mass of literature has been accumulated. We believe that the fact is often lost sight of that such adhesions are principally an effort of nature to effect a cure or to limit infection. The various attempts, hitherto unsuccessful, to avoid such adhesions in inflammatory cases we believe to be efforts in the wrong direction. On the other hand, in clean cases where infection plays no part it is essential that by minimizing handling and exposure of the viscera we avoid the formation of adhesions of no value and possibly of great harm.

In conclusion, we would merely mention the increasing recognition of the importance of abdominal adhesions, and our progress in their diagnosis is especially by the means of the x-rays. A less pleasing subject for our contemplation is our inability effectually to deal with such adhesions when they do exist and cause symptoms.

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DISCUSSION

Dr. John B. Deaver: Without doubt embryology plays a role in the cause of adhesions, but it plays a much more conspicuous role with surgeons with little experience in dealing with peritonitis in all its stages. Those of us operating on many thousands of cases of peritonitis are struck with the fact, brought out by Dr. Ross, that inflammation plays the most conspicuous role. I have seen these adhesions in all stages, from mere strands to organized bands. The type of adhesions originally protected by epithelium disappears as the peritoneum is restored to its normal condition. The type referred to by Dr. Ross and others is the result of destructive desquamation of the epithelium and granulation of the surface of the basement membrane. These ultimately become covered with endothelium but are permanent. I have never been very much impressed by the role played by Jonnescu's membrane or Lane's kinks. Personally my results have been sufficiently satisfactory to warrant my continuing as I have to the present time. Men are governed by their experience.

Dr. Ross (closing): I have nothing to add except to call attention to the fact lost sight of, that the word congenital in connection with these adhesions is not offered in explanation. They are present at birth, but the ordinary conception of congenital is developmental. I do not believe these adhesions are developmental, but inflammatory, although the inflammation occurs in the fetus. There is enough evidence to tell that they are inflammatory, whether before birth or after birth. I regret that I, personally, do not know how to handle this situation. We may let the adhesions alone, operate and cover them over with a raw surface, do an anastomosis, or a resection. I have done all procedures and all have been failures. It is a cause of the greatest amount of chagrin to have these definite entities and not to know how to handle them.

BIRTH-TRAUMATISMS OF THE UPPER EXTREMITY: THE SO-CALLED BIRTH-PALSIES[1]

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In the actual practice of medicine our experience leads us to put the cases which we encounter into various classes. When we start out, almost everything is in the uncertain class. We are not sure of our diagnosis and still more doubtful as to the proper treatment. It requires considerable moral courage to follow the lines which we believe to be right. As, however, the newness of our task wears off we see more clearly, and gradually, as our experience increases, we readily recognize the common complaints and learn how to handle them more or less successfully. No matter, however, how rapidly we advance nor how bright we are, there are continually recurring at various intervals cases which not only puzzle but actually baffle us. The conceit which we have labored earnestly to acquire for so many years collapses and we are left powerless and distressed. We all encounter such cases in our work and we all do what we can to relieve them, but we nevertheless dread their arrival. For me, until comparatively recently, the injuries to the upper extremity commonly called birth-palsies were of that class. In my early days they were regarded solely as true nerve paralyses, and when an infant or child so affected appeared at our orthopedic clinic we, without any compunctions, turned it over bodily to our colleagues, the nerve specialists. They in their turn would turn it over to the physical department for electricity and massage. Sometimes we would dispense with referring them to the neurologist and send

[1] Read March 1, 1916.

them at once to the gymnasium and electrician. I feel I am within the bounds of truth in saying that no one understood them nor intelligently directed their treatment. Even at this time I think you will not find it easy to obtain a clear and generally accepted explanation of the pathology of the injuries, nor are the principles of their treatment definitely established. As active practitioners we may ignore our ignorance of pathology, but when it comes to treatment we cannot dodge our responsibilities. A crippled child is brought to us: we must either help it or condemn it to be left a prey to its infirmity. The continual recurrence of these cases in patients of variable ages convinced me that their treatment in the hands of others was as inefficient as in my own, and stimulated me to see if something could not be done for them. The work has progressed sufficiently to justify me in saying that a great deal can be done to help these unfortunates. I do not wish to claim that the question has been entirely solved, but to present a line of thought in reference to their pathology which leads to a course of treatment which to me, at all events, has been most satisfactory. The condition was recognized by Duchenne in 1872.[1] He described it as an obstetrical paralysis or birth-palsy, and this name has clung to it ever since. He described it as due to an injury of the fifth and sixth cervical roots at their junction.

That Duchenne was at least partly correct is true, but equally true is it that the profession generally appeared to take the view that the affection was solely a paralytic one and therefore more or less incurable, and treated it perfunctorily with routine measures of electricity, massage, and little or no muscular training. The result was that the true principles which underlie the successful handling of the affection were ignored and the results were deplorable. Many of these cases remained seriously crippled all their lives, as many of them do even to this day for the same reason. As time passed Erb described a paralysis occurring from traumatism in adults which was regarded as similar to that described by Duchenne in children. Later Küstner, Whitman, Thomas and

[1] L'electrisation Localise, 3d edition, 1872, pp. 353-367.

Lange, and others directed attention to the articular lesions present and opinions were divided as to the relative parts played by the nerve and articular lesions. To my mind a marked advance has been made by Dr. T. Turner Thomas,[1] who, while not denying the existence of distinct nerve lesions, claimed that the main element of the lesion was articular and proceeded to treat the cases by operative means. Duchenne had already called attention to the existence of a subluxation of the shoulder-joint. Whitman, in 1905, attempted to replace the luxation by manipulation and position, but Thomas resorted to replacement by open operation. The situation is further complicated by Kennedy and Sharp, who resected and sutured the brachial plexus, and Hoffa, Vulpius and Lange, who did an osteotomy of the humerus. While these researches were being carried on I was treating my cases, from the articular stand-point, by conservative measures mostly, and only in a comparatively few recent ones have I resorted to operative procedures. I had long been convinced that an articular lesion was a very important element, and that the most successful treatment must be based on that fact. In this, I believe, Thomas and I fully agree, but I even yet am not convinced that the luxation described by Duchenne and treated by manipulation by Whitman and by operative reduction by Thomas is the essential lesion of the disability. It is true that the treatment directed to the luxation has been of value, but the improvement in function, I feel sure, has been due to the changes produced in the soft parts by the operation rather than to the reduction of the so-called luxation.

My experience in these cases leads me to believe that the condition present consists usually of lesions of the nerves, muscles, and ligaments about the affected parts. The extent and character of the lesions present vary with the mode of production of the original injury and the length of time after birth that the patient comes under observation. If the injury has been severe and the case is seen soon after birth, there may be evidences present of consider-

[1] Annals of Surgery, 1911.

able nerve-paralyses. These will, however, be accompanied by evidences of muscular and ligamentous (articular) lesions also. The signs of paralysis may be so evident as to overshadow the articular lesions, and the existence of these latter then are not suspected. The patient is so young that the *x*-rays may not be of much service, although sometimes bone lesions are shown. The consequence is that treatment is directed only to the nerve lesion, and the articular lesion is untreated. The lesion to the nerves varies greatly in severity. The operations that have been done on the brachial plexus for this affection have demonstrated the fact that absolute rupture of some of the nerves does sometimes take place. This is, however, rare; also some paralyses, particularly of the deltoid, may never disappear, but even this is uncommon. The paralytic symptoms observed, however, are usually due, not to a rupture, but to a contusion or stretching of the nerves, and this is an important fact. If the motor nerves were actually ruptured they practically would not unite and treatment would be futile, but if only contused or stretched, then the probability of recovery is excellent. The tendency is to take an entirely erroneous view of the condition. One is too apt, in a young child, to consider a nerve lesion the prominent factor when the quietude of the arm is due almost or quite entirely to the articular lesions; that is, the capsule and its surrounding ligaments and muscles. It is almost a practical impossibility to determine the exact extent of nerve lesion in young infants. The parts are not moved on account of the articular lesions, and testing the individual muscles by means of the battery is unsatisfactory. Healing of the injuries begins at once and may progress rapidly. If the nerves have not been actually divided, they tend to eventually resume their functions and usually completely recover. It is true that the disability remains, but the essential fact is that this is due, not to the nerve injury which has repaired itself, but to the cicatricial and damaged condition of the periarticular structures. This fact is even yet not generally recognized nor admitted, and, needless to say, the necessity of treating it is ignored. The child then grows up with a crippled arm which may persist through life. In the later years

FIG. 1.—Six-weeks-old infant, showing the typical deformity. The hand is pronated, the wrist flexed (wrist-drop), the elbow slightly flexed, the arm rotated inward and hanging by the side (adduction).

FIG. 2.—Infant with (right) paralyzed arm hanging by its side.

treatment becomes increasingly difficult, owing to the bony deformities which occur, due to the continuous malposition and lack of normal use of the injured member. In young infants the arm will hang lifeless. Usually the fingers can be moved; often there is wrist-drop, lack of pronation and supination and movement of the elbow, and especially of abduction and external rotation of the shoulder (Fig. 1). In older children especially a characteristic attitude is for the arm to hang lifeless by the side (Fig. 2). In some cases the head of the humerus drops away from the acromion process above. The elbow is apt to be slightly flexed and the hand hanging with the dorsum forward. The forearm is in a position of pronation.

Treatment. It is obvious that if we are to achieve satisfactory results in these cases their treatment should be started at the earliest possible moment. The first one who has the opportunity of treating these cases and on whom largely rests the responsibility for their outcome is the physician in attendance. It would seem to be perfectly clear that, if the injury is received at birth, then treatment should be instituted at once. The child is, however, so young and the problem of nourishment so urgent that the local condition will for a time be deliberately ignored, with the intention of attending to it later if it persists. This course may not be so bad for the nerve lesions, but is decidedly harmful for the articular lesions. If the condition is neglected for a few weeks after birth, by that time the injured soft parts will be more or less matted together and the limb will be more or less fixed in the position which it has been allowed to assume. Resumption of function so long as this condition persists is evidently impossible.

The treatment of these cases varies, of course, with the age at which they are encountered. Not infrequently an infant, soon after birth, is noticed not to move one of its arms. It is obviously the duty of the attendant, no matter if the disability does look like a pure paralytic one from injury to the nerves, to make a careful examination of the bones and joints. The caring for a broken humerus or clavicle or injured shoulder or elbow will not militate in the least against the healing of any injured nerves.

At this period of an infant's existence the main object is for it to establish its hold on life, and any measure that handicaps it in this respect is not advisable. It must, however, be borne in mind that even in young infants repair is comparatively rapid, and if an injured part is allowed to remain in an unnatural position for a few weeks, then when treatment is finally instituted it is found that healing has occurred with the parts in their unnatural position, the movements of the arm are abnormally restricted, and the disability is apt to be permanent. The restriction of external rotation of the humerus is marked in these infants only a few weeks old. These injured arms usually hang helplessly by the side and frequently rotate inward, with the palm of the hand backward. This malposition tends to persist, and, if not corrected by treatment, may remain even through life. To correct it the nurse may be instructed to bring the hand across the front of the body, where it may be retained, if necessary, by some simple sling or bandage. She should be shown how, when changing the bandage, to gently pronate and supinate the hand; to flex and extend the elbow; to rotate the humerus outwardly, and, finally, to elevate the arm above the level of the shoulder. The object of performing these passive motions is to prevent the formation of adhesions and contractions which will prevent the performance of these movements in case the muscles regain their power. Massage and electricity are not advised. These measures are to be kept up as the child increases in age, and the restriction of slings and bandages removed as the condition improves.

It is almost impossible to determine how much of the disability present is due to nerve injury and how much to injuries of the bones, joints, and soft parts. We have two means of deciding as to the character of the injury: one is the absence of voluntary movement, and the other is the character of the muscular reaction to the electric current. The presence of restriction of movements a few weeks after birth is proof positive of periarthritic lesions.

The lack of voluntary movement is just as likely to be due to articular as to nerve injury, and the use of the electric current to determine the extent and existence of nerve injury in infants is

extremely unsatisfactory. Even if there is a definite fracture present, it gives one no clue to the extent of other injuries. For these reasons we are practically unable to decide definitely either as to the exact character or the extent of the injuries. It is a fact that in these cases sensation of the affected parts is almost always preserved. This should cause us to be careful in forming opinions, and especially in making prognosis. One should remember that possibly there may be rupture of the nerves and a resultant permanent paralysis, or simply mild nervous and articular lesions followed by quick recovery. Improvement is in many cases rapid, provided treatment is instituted: even if no treatment is given, some of these cases do well; but it is my firm belief that most of the crippled cases that we see later in life are due mainly, not to the character or severity of the original injury, but to lack of treatment. When the child is a few months of age when first seen, then the case is somewhat different. By that time the acute symptoms have disappeared and the arm has become more fixed in its deformed position. It is obvious that the nerve lesions do not cause this fixation, but that it is the articular lesions, and mainly in the ligamentous structures about the joint. While it is true that in some cases injuries to the bones occur, still in the majority evidences of bone injury are absent. This leads us to the belief that the resultant restriction of motion is not due to a bony obstacle, but to contraction or fixation of the soft parts. It follows that before we can expect any improvement in function we must so mobilize the joint that it can exercise its normal range of motion. When this has once been attained, then efforts may be successful in bringing back muscular control. If bony displacements or deposits were the cause of the restriction of motion, then their removal by operative means would be desirable, but if the resistance is due to contracted soft tissues, the persistent stretching efforts may suffice. The restriction of the movements is usually not absolute, but only partial. The arm can be abducted, adducted, and rotated to a certain extent, but not to as great extent as normal. The muscles, therefore, are not truly paralyzed, but can be exercised to an extent sufficient to prevent their atrophy;

but if the joint cannot be moved to its normal extent, the muscles are never fully stretched, and consequently, eventually they will shorten and accommodate themselves to the extent of movement present. If efforts are then made to forcibly enlarge the range of motion, this is resisted, not only by the articular lesions, but by the acquired muscular shortening.

Thus it is seen that the longer the case is allowed to go untreated the more difficult it is to achieve success. These cases a few months old need handling in a trifle more vigorous manner than those seen soon after birth. Like them, the arm is never to be allowed to hang helplessly by the side. The child now is probably in fair general health and is far more active. The bandage or sling is not used so continuously, but the arm is brought forward in a natural position and the infant is encouraged by the mother to use it as much as possible. Evidences of returning power are soon observed, and if objects such as rattles, toys, books, etc. (Fig. 3), are given to the child, it will frequently grasp them with the injured part and make attempts at movements. This should be encouraged by every means possible. These are active movements, but the passive movements are to be faithfully continued. To stretch the parts on the under side of the shoulder-joint the mother should raise the hand high above the head (Fig. 4), also place the hand on the head and slide it down the back of the neck. Above all it is necessary to stretch the anterior part of the capsule. To do this the elbow of the child is kept applied to its side, while with the elbow bent at a right angle the humerus is rotated outward; that is, the forearm is brought from a sagittal to the frontal plane.

This should be persisted in until the forearm points almost directly outward from the side of the body. By comparing its position with that possible with the sound arm of the opposite side the extent of limitation can be determined. To correct any limitation of internal rotation, the elbow may be flexed and the hand placed behind the back. It is obvious that these passive movements should not be made too forcibly, or injury to the bones, especially a fracture of the clavicle or humerus, might possibly

result. I have never seen, however, anything of this sort occur, though I have heard of one such case. There is frequently a tendency to a persistence of a slight degree of flexion at the elbow-

Fig. 3.—Same child as in Fig. 2. After a few weeks of treatment it has begun to hold things in the hand of the affected side.

Fig. 4.—By stretching and exercising this boy can raise the affected arm voluntarily to the extent shown. The bulging of the scapula on the right side shows the contractions on the under side of the joint have not yet been completely overcome. Note that at this age the affected arm is appreciably shorter than the healthy one. Function has markedly improved.

joint. To combat this the forearm should be completely extended, or even put on a splint overnight. To combat the tendency to loss of supination the hand should be frequently rotated or fixed for a time in the position of supination.

Massage is also beneficial, but the value of electricity is doubtful. When the child comes under treatment still later in life, say

FIG. 5.—As a result of treatment this boy's right arm could be rotated outward as far as could his healthy left arm.

between infancy and adolescence, then stronger measures are advisable, and the intelligence of the patient can be to some extent utilized. As before, the first thing is to see that the arm is prevented from hanging to the side in its slightly flexed and pronated position. The same disabilities are present, and of these the lack of abduction and internal rotation are the most marked.

The most efficient means to combat these are to raise the arm

until the elbow is level with or above the shoulder, and to rotate the humerus until the forearm, which at first is pointing directly forward, points upward in the long axis of the body. A plaster-of-Paris bandage or cast is then to be applied around the arm and chest so that their relative position is maintained. The child is then allowed to run around with its arm in this elevated position and its forearm and hand pointing upward, with its ulnar edge forward (Figs. 6 and 7). On subsequent visits the cast is to be

FIG. 6.—Arm and body encased in plaster of Paris. The arm is raised as high as, or higher than, the shoulder, and the humerus is rotated outward, causing the forearm to be vertical.

FIG. 7.—Posterior view. Felt pads are to be wedged in behind the shoulder to push it forward and stretch the soft parts anteriorly.

cut away in front of the shoulder and pads of felt are to be wedged in posteriorly between the shoulder and cast. The effect of this is to push the shoulder forward and to further stretch the soft structures on the anterior part of the joint until the desired correction is obtained.

After a course of several weeks of this treatment the cast is to be removed and the joint subjected to both passive and active movements. The active movements now can take the form of exercises

which can be taught to the child and overseen by the parent. Swinging or suspension from rings is most useful to stretch the under part of the capsule and tends to push the encroaching

FIG. 8.—Young lady, aged twenty years. Arm perfectly useless and held constantly as shown. After operation on the forearm the hand was brought into a position of supination and she could use it in knitting, feeding herself, and many other ways. It ceased to attract attention, and the functional results were extremely satisfactory.

acromion process out of the way. Calisthenics and all sorts of exercises and plays that tend to mobilize the joints are to be encouraged. At this stage frequently there will be absolutely no evidences of nerve-palsy, although there may be considerable

limitation of motion. The object, therefore, is not so much to bring back muscular power as it is to stretch the contracted tissues and perhaps push against and cause the absorption of any obstructing cartilaginous growths. In these young children there is no true bony obstruction, with perhaps the exception in a few cases of an overgrown acromion process. Therefore, if the efforts are persisted in, it is only a question of time until a more or less perfect cure is obtained. As the muscles have practically resumed their functions, the employment of massage and electricity is unnecessary, and reliance is to be placed on active and passive movements.

It is thus seen that the essential part of treatment is first to stretch or manipulate the parts so as to allow them to be placed in their normal positions by passive motion, and then to restore the muscular power by training and exercises so that the patient himself can execute the normal movements by voluntary muscular action. As the patients increase in age fibrous obstructions which restrict motion may be so resistant that it is practically impossible to obtain the extent of motion desired. Also, the persistent abnormal position of the limb may result in a distortion or abnormal direction or condition of the bones. If bones are not used normally they will not grow normally. As a consequence it is desirable to attempt to attain the desired object by operative means. The exact operative measures which are of service will, of course, vary with the individual cases, and as yet they have not been sufficiently tried out to enable us to standardize them.

There have been many operations done with the object of reducing the so-called luxations often seen in these cases. Notwithstanding the apparent improvement which has followed such attempted repositions, I have not as yet seen my way clear to advocate them. What have been termed luxations are, to my mind, subluxations, and the malposition in itself seems hardly to be of sufficient extent to either account for the disability present or to justify us to expect much functional improvement by removing it. The good result which has followed them I regard as being due, not to the replacement of the head of the humerus, but rather to the free division of restraining tissues and the placing of the parts in a better position.

As has been already stated, in the shoulder-joint the two motions which are restricted are abduction and external rotation. It is probably true that in some cases the persistent falling away of the head of the humerus from the acromion process has favored an overgrowth of that process so that it interferes with full abduction. In such cases the removal of a part of the obstructing acromial process may be of service. I feel, however, that the greatest obstacle to abduction comes from contraction of the fibrous structures on the under side of the joint rather than the upper side; also, that if obstruction is offered by the acromion above, it is only when the arm is brought into extreme abduction and the limitation of movement so caused is but slight and readily compensated for by a slight increase in the range of rotation of the scapula. The operative procedure which I prefer in these cases is as follows:

An incision is made anteriorly through the edge of the deltoid muscle. This avoids the cephalic vein and brings one down immediately over the head and upper end of the humerus. The deltoid is parted in the direction of its fibre and widely retracted. The humerus is then rotated outward, and this makes the subscapularis tendon tense and carries the long tendon of the biceps muscle to the outer side. The capsule and tendon of the subscapularis are then divided from above downward, separating them from their attachment to the lesser tuberosity. This will allow the humerus to be rotated farther outward. If the tendon of the pectoralis major interferes with either external rotation or abduction, it, too, may be divided. If abduction is not sufficiently free, the attachment of the capsule at the under side of the joint may be divided by keeping the knife close to the bony surface of the humerus. The skin incision is then to be closed and the arm placed in marked abduction, with the humerus outwardly rotated, in a plaster-of-Paris dressing. The subsequent treatment is the same as that employed for non-operated cases.

Of the value of this operation I have no doubt. In the two cases in which I have performed it marked improvement has been obtained. It is neither a dangerous nor crippling procedure, and

the worst that can be said about it is that it may not succeed, but, even if such is the case, the future of the patient has been made no worse. In the very bad cases it should be given a trial. As yet I have not thought it advisable to operate on the elbow for the sake of increasing flexion or extension. In one case, aged about twenty years, supination was impossible (Fig. 7). In this case the pronator teres muscle became tense when supination was attempted, therefore an incision was made over the middle of the radius and the insertion of the tendon divided. This allowed an additional degree of supination, but not enough. Another incision was then made over the head of the radius, the external lateral and orbicular ligaments were divided, and the tissues detached for a short distance down the inner side of the neck and shaft. On supinating the hand, the head of the radius rose directly out of its bed and projected forward. It was evident that the radius in its growth had become twisted, so that when supination was performed the head was thrust forward. Therefore the head and neck were removed nearly or quite down to the insertion of the biceps muscle at the radial tubercle. It is needless to say that the greatest care is to be taken not to wound the posterior interosseous nerve which runs through the supinator (brevis) muscle, or wrist-drop will be caused. The wounds being closed, the forearm was dressed on a splint, with the hand in the position of extreme supination.

This operation has only been done in one case, but the results were so extremely satisfactory as to justify me in urging a trial of it in suitable cases. These cases of injuries at birth are fairly numerous in the community, and the question suggests itself whether or not some of them could not have been prevented. It is possible that physicians and midwives may have an exaggerated idea of the amount of traumatism that a child may be subjected to at birth without serious injury. At all events it may not be out of place to call attention to the fact that the attendant is to a certain extent responsible for these injuries, and the greatest care should be taken to avoid them.

The profession also needs to direct their attention to this class

of cases, and not neglect them as heretofore. There is no doubt that the large number of dependent cripples in our community is mostly composed of those whose deformity could either have been prevented from arising or remedied after its existence had become manifest. Now that we understand these birth injuries better and can do so much for them, the profession should cease to ignore them and rise to their duty of seeing that they receive efficient treatment.

Conclusions. 1. There is primarily an injury of both the nerves and articular structures.

2. In a few cases some of the nerve-paralyses are permanent, but in most cases recovery of the nerve lesions ensues and the paralysis disappears.

3. In most of the cases, especially after the lapse of a year, the disability is due to restriction of normal motion by contracted periarticular structures.

4. The disabilities are five in number: (*a*) wrist-drop; (*b*) lack of supination; (*c*) lack of complete elbow extension; (*d*) lack of complete external rotation of the humerus; (*e*) lack of proper abduction.

5. They are to be treated by increasing the range of motion—by operation, by stretching, by splints and fixed (plaster-of-Paris) dressings; by persistent exercises and physical education.

6. Even after the part can be placed in a normal position by passive means, a long period of exercise and education is necessary before proper control and use are acquired.

7. Most of these cases can be greatly improved or practically cured, and the presence of disability in after-life is usually the result of previous neglect.

8. As recovery of the nerve lesion nearly always ensues, operations to reunite them are inadvisable for a long period after birth, perhaps several years.

DISCUSSION

Dr. T. Turner Thomas: Of course I am very much interested in this subject, and interested in seeing Dr. Davis take this view of this question. It has been only a few years since there was but one view of the etiology of these cases. It was uniformly believed that all were due to injuries of the brachial plexus. Today many believe them due to injuries of the skeleton in the shoulder region. We are getting back to the position in which Duchenne was when he first began his work on the subject.

Dr. A. P. C. Ashhurst: I think that Dr. Davis has done very well to bring before the College a subject such as this, which interests so many of the specialists in the profession, the obstetrician, the family physician, the pediatrician, and the neurologist and the orthopedist, as well as the general surgeon. In addition to all the patients seen with Dr. Davis in his clinic, and just to show how frequent the condition is, I may say that I have had under my care within the last two or three years nearly 40 cases.[1] I do not know in what proportion of this number dislocation of the shoulder has been present, but it is a large number. It was the dislocation of the shoulder to which Dr. T. T. Thomas called particular attention, which first aroused my interest and gave a clue to the best method of treatment. I had one case, a child, aged three months, in whom a posterior dislocation was present, which was confirmed by the *x*-rays. I felt that there was no reason why the case should not be treated as we would treat the same condition in the hip. Therefore a few months later I gave ether and reduced the dislocation and held the shoulder in a plaster dressing in abduction and external rotation as in the hip. From that time on we knew pretty well what to do with cases of dislocations. But dislocations are not the only lesions which occur, and the dispute at present is whether the dislocation caused by the original injury at birth, or follows a traumatic paralysis of some of the shoulder muscles. I had one patient under my care in whom at birth the arm was elevated, the forearm held across the front of the neck, and a posterior dislocation was present. The arm flew back into the elevated position if it was not held down on the chest. So there is no question that dislocation does occur at birth. These cases used to be called congenital dislocations of the shoulder. There are also cases in which at the first examination no dislocation is present, but in which after a few months it

[1] These will be reported in detail by my assistant, Dr. A. Bruce Gill.

appears. I have had at least two such cases. There are also cases of dislocation in which there is a nerve lesion. Not only will be seen reactions of degeneration, which can be detected with more or less accuracy in the first year of life, but also I have had one patient who at three years of age was still anesthetic and powerless throughout the entire upper extremity, and I thought the case hopeless. Later I sent for this child again, and although three years of age, motion and sensation, though not good, were beginning to be apparent. I found upon further examination that there was dislocation of the shoulder. But as Dr. Davis and Dr. Thomas have said, lesions of the nerves in most cases are of secondary importance.

In treatment I have come to the view that the dislocation should be reduced. Dr. Davis seems to ignore that, but treats the soft parts and lets the dislocation alone. Up to the age of three years I have been able to secure permanent reduction without open operation, but those over this age reduced bloodlessly have recurred. None of the cases in which I have operated (bloody reduction), 5 in all, have recurred. The ages of these patients varied from five to sixteen years. In all I used the approach to the shoulder-joint described by Kocher with Senn's modification of the skin incision. I found it impossible to reduce the shoulder completely until after division of the tendon of the subscapularis muscle. To keep the shoulder externally rotated, I plicated the tendons of the supraspinatus and infraspinatus at their insertion into the greater tuberosity. In my oldest patient after I had done this I was obliged to cut off part of the acromion before it would go back where it belonged. In other patients past the age where bloodless methods are satisfactory, and in whom there was disability from contractures of the soft parts without posterior dislocation, I have gone in from the front and divided the subscapularis and pectoralis major. In all cases the shoulder is kept in full abduction and external rotation in plaster of Paris for at least three months.

I would conclude, then (1) that if these patients are treated from the time of birth, as Dr. Davis has emphasized, and in the way he advises, most will recover with little permanent disability; (2) that in a few of these cases at a later period and in almost all seen for the first time a number of months after birth (especially if dislocation of the shoulder is present) it will be necessary to treat the upper extremity in plaster of Paris in abduction and external rotation for a period of not less than three months; (3) that where dislocation is present this should be reduced, if necessary under anesthesia, as soon as practicable after it is recognized; (4) that in patients over three years of age bloodless reposition seldom will be permanently successful; (5) that operative treatment must secure

anatomical reposition of the humerus in the glenoid, with restoration of free external rotation and abduction, which should be maintained by fixed dressings for a period of not less than three months; (6) that the older the patient when first subjected to surgical treatment the more difficult it is to secure a return of normal function, owing to bony deformation not only at the shoulder, but in some cases also at the elbow and the wrist. (7) and finally that in the vast majority of cases nerve lesions are insignificant and do not require any special treatment.

Dr. Davis (closing): The whole subject is in a more or less unsettled stage regarding both pathology and treatment. We are sure, however, that today a great deal can be done for these cases. In spite of the fact that some of these cases shown are not brilliant from a demonstrative stand-point, still from an utilitarian stand-point and increase of function of the part they were well worth the trouble expended on them.

THE USE OF THE "KARELL CURE" IN THE TREATMENT OF CARDIAC, RENAL, AND HEPATIC DROPSIES[1]

By EDWARD HARRIS GOODMAN, M.D.

The various measures recommended for the successful treatment of failing cardiac, renal, and hepatic functions, with their concurrent edema of more or less severity, are legion, and one would be unwise to undertake the treatment of such conditions without a full appreciation of the value of each. Drugs, physical therapeutics, and diet form the triad upon which reliance is usually placed, and generally speaking, in the lay as well as in the professional mind, the greatest of these is drugs. There are many dropsical cases in the treatment of which one must use all three; there are many where physical measures may be safely dispensed with, and there are many cases of severe renal and cardiac breakdown in which drugs as well as physical measures may be disregarded, but there are none where diet has not earned a well-deserved fixed place.

It may be stated, without fear of serious criticism, that the majority of cardiac dropsies and a large proportion of dropsies of renal origin will improve with the combination of rest in bed and an appropriate diet. The diet which has served me best, and which I have employed successfully for the past seven years, is that known as the Karell diet, or the Karell cure. Although half a century has elapsed since Karell published his paper (1866), this particular form of diet bearing his name seems to be but little known and but rarely used. In Germany it is slowly finding its place, but as recently as 1908 Jacobs (*München. med. Wchnschr.*, 1908, p. 839) wrote that he could find no mention of it in the text-books, and

[1] Read April 5, 1916

but little practical knowledge of its existence among physicians. In America it is known, but not intimately, and it has not secured for it the acclaim which would be its portion were it employed more frequently.

During my association with Dr. John H. Musser in the Presbyterian and University Hospitals, and with Dr. James E. Talley in the former institution, I have been given the opportunity to make free use of this method of treatment during the past few years, and have employed this diet in many cases, between a hundred and a hundred and fifty approximately. This experience, which has been productive of improvement in the majority of these patients, is the *raison d'être* of this paper.

The technic of the Karell cure is simple and easily carried out so far as the physician is concerned. The patient receives daily at 8 and 12 A.M., and 4 and 8 P.M., 200 c.c. of raw or boiled milk, warm or cold, according to taste. No other food or liquid should be given. This strict diet sometimes, nay many times, meets with violent opposition from the patient, and great complaint is made because of thirst. Thirst is particularly tormenting during the first three or four days of the "cure," and oftentimes it becomes necessary to allow the patient to rinse out his mouth with water, instructing him to swallow none, however. Hunger is not so common a complaint, but when urgent a small piece of dry toast or zwieback may be given with each portion of milk. During the first few days the patient requires continual encouragement to persist with the treatment, but the moral effect of the rapid loss of weight, as shown by daily weighings, together with the very evident decrease of the edema, prove sufficient argument to him, and no further complaint is heard. Just how long this very strict diet is to be continued depends on the rapidity with which edema diminishes, and on the patient's plea for more food. Usually the diet may be increased at the end of a week's time by giving a soft-boiled egg (without salt or pepper) at 10 A.M., and a piece of zwieback at 6 P.M. The next day an egg may be given at 10 A.M., and 2 P.M., with a piece of white bread, and from now on food is gradually increased until a full diet is being taken. During

this time the daily quantum of liquid should not exceed 800 c.c., and this fluid should be in the form of milk until the patient receives a full diet, when the milk may be discontinued and cocoa or tea substituted, the amount of liquid remaining the same, however. No more than 800 c.c. of fluid should be taken for from 2 to 4 weeks after the disappearance of edema.

The "full diet" spoken of above is a misnomer, for the diet should be salt-poor, and the following foods comprise this dietary: meat, fresh-water fish, cream soups, fresh eggs cooked in any form or raw, rice, corn, hominy, Indian corn, endive, peas, string beans, French beans, artichokes, onions, leeks, carrots, salads except lettuce, cauliflower, potatoes, cereals of all kinds, butter (unsalted), bread without salt, sweetmeats, sugar, chocolates, cocoa, tea, coffee, fruits, milk, cream cheese and Swiss cheese, puddings, junket, ice-cream.

During the "cure," which it seems needless to say must be carried out with the patient in bed, the bowels should be kept open, and for this purpose laxatives in pill form are preferable to salines merely because they require no water for their administration. In typical cases, that is, in those individuals who begin to lose weight through the loss of edema and who show increased diuresis, no other medication is required; but when improvement is not seen at the end of three days, or when symptoms are urgent (dyspnea, oppression, restlessness, unduly frequent and weak pulse, active uremic signs), the drugs and other measures usually indicated in such conditions should be used. This question will be further discussed later.

The article by Karell in which he described his milk cure was published in *Archives générale de Médecine*, 1866, viii, 513, and apparently was entirely forgotten, and would, no doubt, have remained in obscurity, had not Jacob (*München. med. Wchnschr.*, 1908, p. 839) reported the results obtained with it in Lenhartz's clinic, where it had been used for about fifteen years. Just how many of the few writers on the subject have read Karell's original paper it is impossible to say, but Jacob has given the year of publication as 1868 instead of 1866, and his error has found its

way into practically every article since 1908, so that the inference that few have read the original is perhaps justified. Possessed as it is of great practical interest and of historical interest as well, written in a peculiarly intimate manner and naïve in spirit, Karell's paper should be read at first hand, but I cannot forbear quoting at length from this very interesting article. He writes:

"It is always with a rich profusion of remedies and with the greatest confidence in their efficacy that the medical youth enters upon the practical exercise of his profession. However, experience does not long delay in demonstrating the inanity of that pretended richness; the circle of medicines prescribed narrows, year by year, and the physician whose age has ripened talent finally finds himself obliged to confess that the surface of his finger-nail would be large enough upon which to inscribe the names of the medicines used in practice.

"If I confess to something of the same nature, if at the end of thirty-four years of practice a sort of skepticism takes possession of my spirit relative to the curative virtue of many medicaments, I have, on the other hand, gained faith in the efficacy of certain means whose object is to change and to modify nutrition.

"If I call attention today to the methodical cure by means of milk, I do it in the firm conviction that the number of cures brought about by this treatment are due, in large part, to the judicious use and to the strict observance of the method. It is only thus that truly startling results have been obtained, results which the general public have qualified as marvelous.

"It may be objected that milk, as a remedy, is fairly well known and that every physician uses it according to the exigency of the case. I confess that all physicians are sufficiently informed regarding the virtue of milk as food and as antidote, but I speak from experience when I say that, in general, the milk cure scrupulously administered in amounts rigorously fixed is not sufficiently or but rarely recognized by practitioners as a heroic and sovereign remedy."

Karell, who was physician to the Emperor of Russia, reminisces on his excursion with the monarch through Russia, and speaks of

his pleasure in seeing the milk cure being used extensively in many cases. He then transcribes a letter from the famous Niemeyer, which is worthy of being again transcribed. After having communicated to the Tübingen professor his experiences, he received the following letter some time later:

"I am infinitely grateful to you for having recommended the milk cure; I have used it so often and extol it in such a way that it would make you smile. If one were sincere enough to recognize that there exists a very large number of diseases whose cause should not be sought in disturbances of certain organs, but rather in a perverse state of nutrition, of which we know neither the extent nor the nature, he would believe in the curative virtue of milk and would regard it as a true scientific advance that one can find in that article of food a harmless measure and at the same time one so efficacious in changing completely the state of nutrition."

Karell used the milk cure in at least 200 cases and found that it gave good results when other measures proved to be valueless. As to the principle underlying its virtues he declines to give any opinion, and this he defends by claiming: "The art of healing would surely remain sterile if one should limit himself only to those remedies whose effects can be controlled even to the smallest details." The diseases which are treated best by the cure are particularly dropsical conditions, but he gives a large list of other diseases of more or less chronic nature.

The writer has restricted himself to the employment of the Karell cure in cases of renal, cardiac, and hepatic edema, and it is only these diseases which will be discussed in this paper, although other clinicians have used it in bronchitis, emphysema, gastro-intestinal conditions, and particularly in the treatment of obesity. Edema, from whatever cause, is the condition *par excellence* in which the most notable results are achieved, however.

ILLUSTRATIVE CASES

The following cases will illustrate the effect of the Karell cure in cases of edema. The first three charts exhibit only loss of weight.

Chart IV is that of the patient Case III on his first visit to the hospital, and shows the rapid increase of diuresis, then a falling off in the amount of urine, which provided an indication for the use of other therapeutic measures. Charts V and VI illustrate loss of weight and increase in diuresis (delayed in Case VI). Chart VII shows marked diuresis amounting to 531 ounces in four days with unusually rapid loss of weight. Chart VIII illustrates the effect of the milk diet in a case of hepatic cirrhosis. Chart IX illustrates failure of the Karell cure, slow and halting loss of weight, and very small amounts of urine, the highest amount being but thirty ounces. Chart X is devised chiefly to show the lowering of blood-pressure, both systolic and diastolic due to the Karell diet. Chart XI shows retention of chlorides on a salt-poor diet. Prompt elimination of chlorides and increased diuresis on Karell cure plus caffein and hot packs. Chart XII contains curves of body weight, chlorides, and urine.

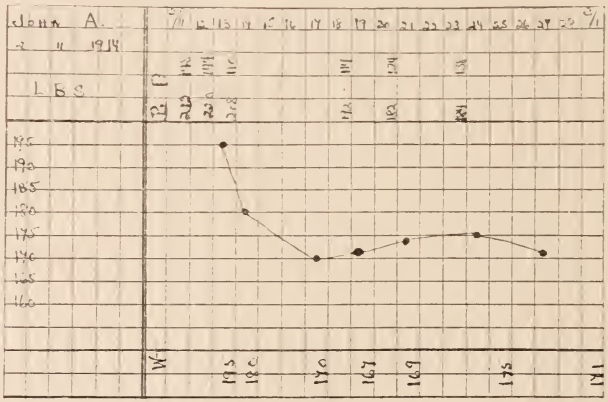

Chart I[1]

Case I.—Diagnosis: Endocarditis; arteriosclerosis.

John A., aged seventy years. Admitted February 11, 1914, complaining of dyspnea and edema of feet.

[1] Acknowledgement is made to the Archives of Internal Medicine for the use of these charts.

Present Illness. Patient states he was fairly well until three years ago, when he became dyspneic and feet began to swell. Since this time dyspnea has been pronounced on exertion; he has marked palpitation of the heart.

Examination. Well-nourished adult white male. Heart enlarged to right and to left. Systolic murmur at apex, systolic murmur at aortic cartilage. Extrasystoles. Vessels sclerotic. Legs greatly swollen.

Urine: 1020; trace of albumin; no casts.

Blood: Hemoglobin, 75 per cent.; leukocytes, 10,850; red blood cells 4,970,000; blood-pressure, 212 to 148.

Result of treatment: Patient is fairly good condition when discharged, March 4, 1914. Edema disappeared entirely. Subjective symptoms much improved. Some reduction in blood-pressure.

Note. Chart shows loss of weight of 26 pounds in ten days.

Case II.—Diagnosis: Aortic insufficiency; intermittent heart-block.

Frank P., aged thirty-nine years. Admitted November 7, 1913, complaining of cardiac palpitation, dyspnea, and edema of leg and scrotum.

Present Illness. Patient was well until two months ago, when, on returning home from work, he suddenly became very dyspneic. Similar attacks were of frequent occurrence after this.

Previous Medical History. Contracted lues and Neisserian infection at twenty-eight years of age. Was treated in Presbyterian Hospital two weeks ago for condition similar to the present illness.

Examination. Poorly nourished adult male. Marked pallor and cyanosis. Dyspnea pronounced. Edema of legs and genitalia. Heart, apex beat is in sixth interspace, 2 cm. outside left midclavicular line. Right border at right parasternal line. Left border 3 cm. to left of left midclavicular line. Systolic thrill is felt at apex. Apex beat fluttering and diffuse. Presystolic murmur at apex, followed by short diastolic murmur. Double murmur at aortic and pulmonary cartilages. Liver palpable. Lower extrem-

ities edematous, also penis and scrotum. Pulse irregular, rapid; arteries sclerosed. While in ward typical attack of Stokes-Adams syndrome.

Urine: 1015; cloud of albumin, no casts.

Blood: Hemoglobin, 58 per cent.; red blood cells, 3,280,000; leukocytes, 6400; blood-pressure 205 to 115.

Result of treatment: Loss of weight 8 pounds in seventeen days. Nine days after beginning Karell cure edema had almost entirely disappeared; there had been a loss of weight of 13 pounds. Subjective symptoms improved, no sharp attacks of dyspnea such

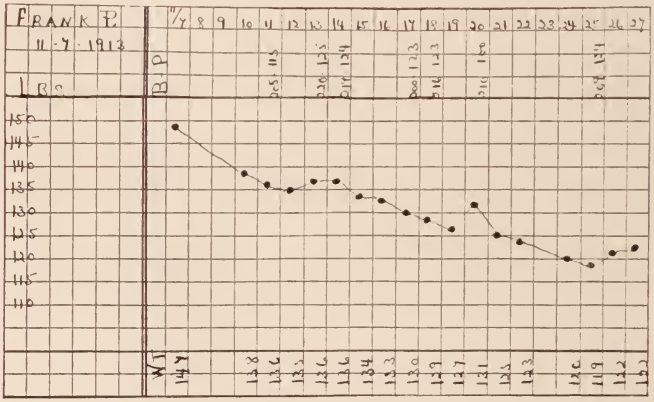

Chart II

as were complained of on admission, although patient is still dyspneic. Heart is more regular, pulse less frequent, no change in blood-pressure. Patient discharged November 28, 1913, much improved.

Note. Chart shows loss of weight only. Urine was not saved quantitatively, so this is not recorded. Prognosis in this case was grave, although there was benefit from the Karell cure. Patient returned December 29, 1913, with symptoms similar to those on previous admissions. The Karell cure was again prescribed, but there was no improvement. Cardiac stimulants were employed, but patient succumbed January 6, 1914.

Case III.—Diagnosis: Cardiorenal disease.

Frederick P., aged sixty-three years. This patient who is the same as Case IV, was readmitted to the hospital November 30, 1915. He had been coming to my dispensary since his discharge, but the exertion proved to be too much, and ten days after his discharge dyspnea became marked; legs, penis, and scrotum also became edematous. The condition on admission was the same as that sketched above.

Result of treatment: Loss of weight amounted to 22 pounds in 13 days. There was again disappearance of all edema and great improvement in subjective symptoms.

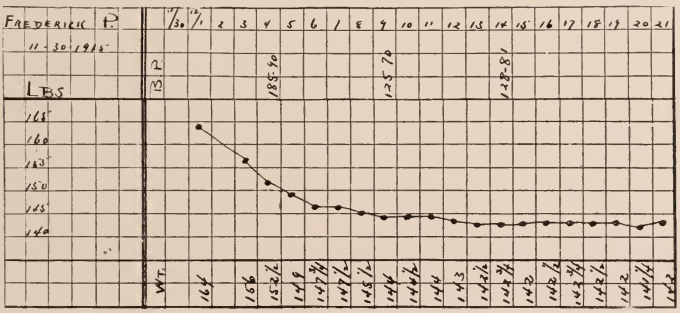

Chart III

Note. Chart reproduced merely to show loss of weight. No accurate record was kept of the amount of urine, as the patient stipulated before entering the hospital the second time that he be allowed to go to the toilet room, so the urine could not be collected quantitatively, as was possible during the patient's first visit to the hospital.

Case IV.—Diagnosis: Cardiorenal disease.

Frederick P., aged sixty three years. Admitted October 9, 1915, complaining of retention of urine and shortness of breath.

Present Illness. For two months patient has had a smothering feeling at night, causing him to awaken with a start. He has observed that he became dyspneic on the slightest exertion, and would, when walking, have to stop and rest after going the dis-

tance of a few hundred feet. Has had no polyuria and no other symptoms worth noting, except that since September 3, 1915, a month previous to admission, he has become very weak.

Previous Medical History. Has been a sufferer from asthma, and a year ago had nasal operation which failed to furnish relief. Other history negative.

Examination. Well-built man, complexion pasty. Dorsal decubitus, but the slightest exertion causes dyspnea. Face edematous. Some hydrothorax on both sides. Heart apex in sixth

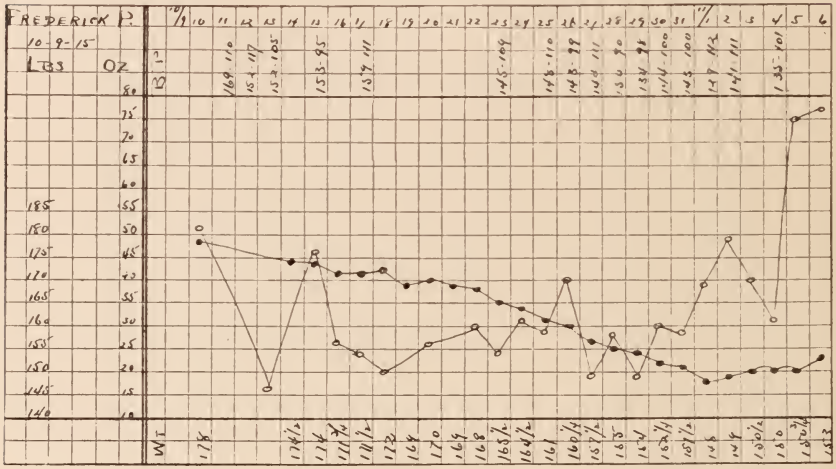

Chart IV

interspace to left of midclavicular line. Marked pulsation in fourth and fifth intercostal spaces. Right border 4 cm. to right of midsternal line. Left border 15 cm. to left of midsternal line. No murmurs at apex, sounds are weak. Second sound at pulmonary and aortic cartilages accentuated. Marked edema of entire body, abdomen shows ascites. Legs, scrotum, and penis very edematous. Arteries sclerotic. Catheterization required. Eye-grounds negative.

Urine: 1013; albumin, faint trace; hyaline and granular casts. P. SP. 7 per cent.

Blood: Hemoglobin, 84 per cent.; red blood cells, 2,450,000; leukocytes, 5350; blood-pressure, 169 to 110.

Result of treatment: Disappearance of edema. Improvement of subjective symptoms. Loss of weight gradual but amounting to 30 pounds in thirty days. Reduction of blood-pressure. In this case hot packs were used and caffeine 0.36 gram a day. Patient discharged November 6, 1915.

Note. Chart shows marked increase in amount of urine with later a falling off in quantity. On the 12th, because of this, hot packs and caffein were ordered. Later digitalis (2 c.c. tincture daily).

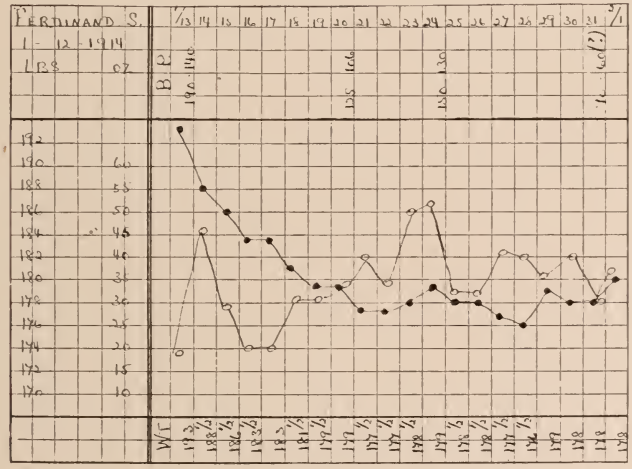

Chart V

Case V.—Diagnosis: Cardiorenal disease.

Ferdinand S., aged sixty-one years. Admitted January 12, 1914, complaining of dyspnea and swelling of lower extremities.

Present Illness. Began six months ago with shortness of breath, dizziness, and swelling of feet and legs. Since then these symptoms have become intensified, until now patient can scarcely get his breath when he makes even the slightest exertion.

Previous Medical History. Pneumonia ten years ago and acute articular rheumatism two years ago.

Examination. Well-nourished elderly white male. Marked dyspnea. Lungs, chronic passive congestion at bases. Heart sounds weak but regular. No murmurs. Slight enlargement to left. Ascites. Liver palpable. Lower extremities edematous.

Urine: 1029; trace of albumin; few hyaline and granular casts at times.

Blood: Hemoglobin, 70 per cent.; red blood cells 4,800,000; leukocytes, 7950; blood-pressure, 190 to 140.

Result of treatment. Fall in body weight and gradual increase in diuresis. Until January 19, 1914, patient felt no better, but at this time he was improved, and the blood-pressure, body weight, and edema were all diminished. Patient discharged February 10, 1914, much improved.

Note. Chart shows drop in body weight. Sudden diuresis followed by fall (urine was not accurately measured for forty-eight hours, January 16 and January 17). Then again increased amount. Gradual fall in blood-pressure.

Case VI.—Diagnosis: Chronic nephritis.

Benedict P., aged twenty-eight years. Admitted July 10, 1913, complaining of dyspnea, distention of abdomen, edema of legs.

Present Illness. Six months ago patient noticed that feet were swollen. Unable to walk without becoming much fatigued. Two weeks ago edema became worse and dyspnea appeared.

Examination. Nutrition good. Anasarca, ascites. Edema of lungs. Heart; apex in fifth interspace midclavicular line, right border indefinite, left border in left midclavicular line. Sounds are indistinct. No murmurs. No enlargement of spleen or liver. Eye-grounds negative.

Urine: 1018; heavy cloud of albumin; hyaline and granular casts; phenolsulphonephthalein, 18 per cent.

Blood: Hemoglobin 53 per cent.; red blood cells 2,680,00; leukocytes, 9900; blood-pressure 129 to 80.

Result of treatment. Loss of weight, 26 pounds in fourteen days. Slight increase in urine excretion. Edema much less,

although not entirely gone on discharge. Disappearance of all subjective symptoms. Marked increase in chloride output. Discharged August 27, 1913, much improved.

Note. Chart designed to show loss of weight and delayed diuresis.

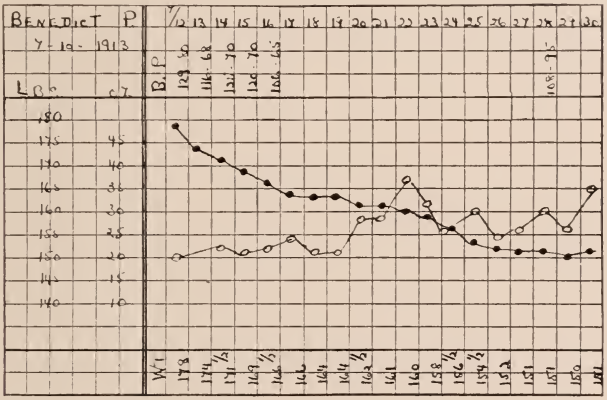

CHART VI

CASE VII.—Diagnosis: Cardiorenal disease.

Mary S., aged fifty-seven years. Admitted October 7, 1914, complaining of shortness of breath, swelling of legs and distention of abdomen.

Present Illness. Patient has been suffering with edema of the legs for about a year or more, but she became worse about five weeks ago. Began with abdominal distention, then dyspnea appeared and became so pronounced that she could not lie down. She has been forced to sleep in her chair. Palpitation of heart marked. Blood-tinged expectoration.

Previous Medical History. Rheumatism at age of sixteen years.

Examination. Patient fairly well nourished, but is very edematous. Pallor. Heart irregular, enlargement to left and to right, systolic murmur at the base. Arteries sclerotic. Liver palpable; ascites. Marked edema of legs.

Urine. 1016; albumin, trace; hyaline and granular casts.

Blood: Hemoglobin, 60; red blood cells, 3,640,000; leukocytes, 7600; blood-pressure, 145 to 90.

Result of treatment. Disappearance of all edema. Loss of weight, 54 pounds in ten days. Reduction in blood-pressure from 145 to 90 to 125 to 80 after seven days.

Note. Chart shows rapid loss of weight. On second day of treatment urine was 100 ounces, on third day 148, fourth day 138, and fifth day 145 ounces or a total of almost 16 liters (15,940 c.c.) in five days.

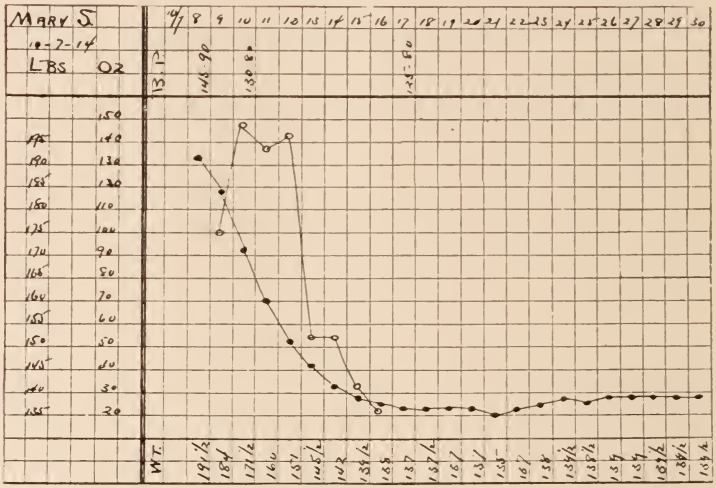

CHART VII

CASE VIII.—Diagnosis: Hepatic cirrhosis.

Edward R., aged thirty-five years. Admitted January 12, 1914, complaining of "asthma," dyspnea, swelling of feet, legs, and abdomen.

Present Illness. Has had shortness of breath for a number of years, but during the past six weeks he has been worse, and is now forced to rest sitting up. Cough has become very troublesome.

Previous Medical History. With the exception of asthma patient has never been sick, but was in the University Hospital

in 1905 for some gastric trouble. Has always taken alcohol to excess; admits Neisserian infection, but denies lues.

Examination. Large, well-built man. Semirecumbent decubitus. Breathing labored and wheezing. Pallor. Lungs emphysematous, sibilant rales. Heart, apex beat not visible, sounds weak, soft systolic murmur at apex. Area not enlarged. Abdomen-ascites. Liver palpable. Edema of feet and legs.

Urine: Negative.

P. SP., 66 per cent.

Gastric analysis: Total acid, 8; free HCl, 9.

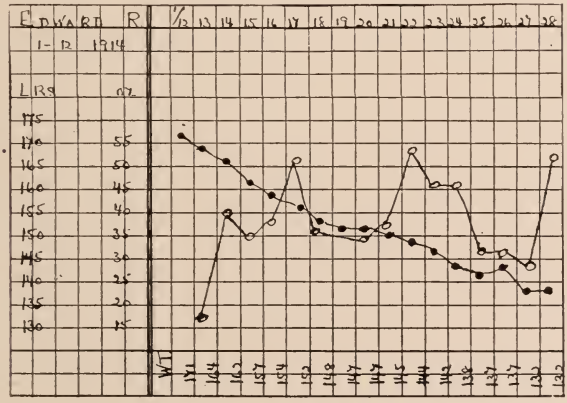

CHART VIII

Stools: Occult blood present.

Blood: Hemoglobin, 45 per cent.; red blood cells, 3,390,000; leukocytes, 10,650; blood-pressure, 120 to 70.

Result of treatment. Loss of weight, 39 pounds in sixteen days. Diet was increased on seventh day of treatment. Patient improved when discharged, February 26, 1914.

Note. Chart shows rapid loss of weight and prompt increase in amount of urine.

CASE IX.—Diagnosis: Endocarditis; decompensation.

William W., aged twenty-two years. Admitted November 4, 1913, complaining of dyspnea, cough, and expectoration; pain on inspiration; scanty urination; swelling of feet; ascites.

Present Illness. Has never been sick with similar illness. He was apparently well and walking about until two weeks ago, when he began to notice swelling of the feet and began to lose his appetite. Also developed a severe pain in the right infraclavicular region, sharp, stabbing in character, made worse by taking a deep breath, and on coughing, which soon developed. Became dyspneic on exertion. For the past two weeks has been passing but little urine, which was dark in color. Pain in back over renal region. Face started to swell about two weeks ago, and about this time noticed that his abdomen became swollen.

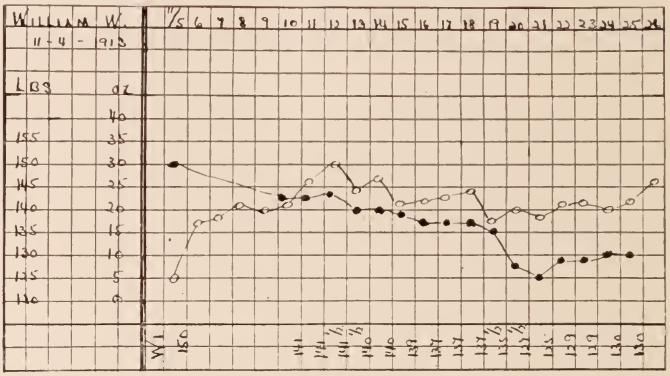

CHART IX

Previous Medical History. Negative. Used alcohol to excess and does hard work.

Examination. Well-nourished young male. Uremic odor to breath. Face swollen and flushed. Dyspneic. Abdomen swollen. Lungs tuberculous, pleural effusion. Heart, apex beat in sixth interspace, 14 cm. to left of midsternal line. Upper border of third rib, right border 4½ cm., left border 14 cm. from midsternum. Sounds are irregular, gallop rhythm. Systolic murmur at apex. Abdomen shows ascites. Liver palpable and pulsating. Lower and upper extremities edematous.

Urine: 1080; heavy cloud of albumin; no casts.

Blood: Red blood cells, 4,710,000; leukocytes, 18,150; blood-pressures, 131 to 93.

Result of treatment. On November 10 patient seemed a little better, was not so dyspneic, but began to be jaundiced. Later paracentesis thoracis (November 19, 1913). Patient gradually became worse, and despite active use of cardiac stimulants, succumbed on November 28, 1913.

Note. Chart shows only moderate loss of weight, very slowly achieved. Urine amounts continually low. No improvement in pulse rate. Prognosis guarded on admission.

Case X.—Diagnosis: Chronic nephritis.

James K., aged forty-nine years. Admitted September 2, 1913, complaining of swelling of legs, genitalia, and abdomen.

Present Illness. Patient was well until five weeks ago, when he noticed that he was beginning to tire easily; had general malaise and was losing appetite. One week ago he first observed a swelling in the face and the next day general swelling throughout body.

Had diarrhea one week ago; soreness across abdomen; pain in back; polyuria. Three weeks ago had pains about the heart which lasted for three days and prevented him from taking a full breath. Dyspnea and palpitation also complained of.

Previous Medical History. Had nephritis with dropsy eighteen years ago. Treated in Presbyterian Hospital and was discharged as cured.

Examination. Well-nourished white male adult. Slight cyanosis. Face puffy. Breath urinous. Few moist rales at bases of both lungs. Heart, right border, 2 cm.; left border, 12 cm.; upper border at third rib, first sound at mitral area, roughened and prolonged. Pulse regular. Arteries sclerotic. Many hyaline casts. Edema general.

Urine: 1010; heavy cloud of albumin; many hyaline casts; phenolsulphonephthalein 40 per cent.

Blood: Hemoglobin, 60 per cent.; red blood cells, 4,570,000; leukocytes, 11,200; blood-pressure, 175 to 92.

Result of treatment. Loss of 16 pounds of edema in eight days, with disappearance of all swelling. Diuresis increased, but not

charted, as amount was not accurately measured. Gradual fall in blood-pressure, systolic and diastolic. Disappearance of all subjective symptoms. Discharged September 17, 1913, much improved.

Note. Chart designed to show loss of weight and decrease of blood-pressure.

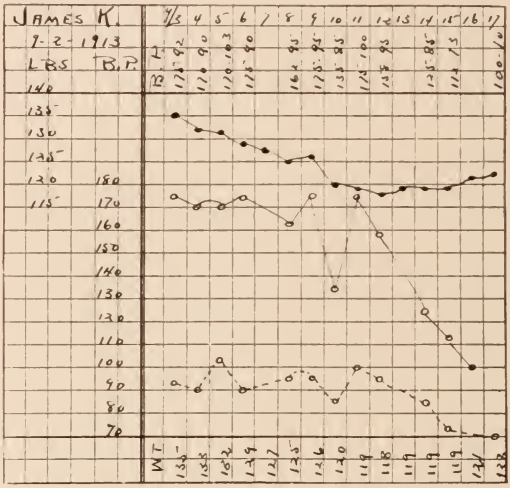

CHART X

CASE XI.—Diagnosis: Nephritis.

Margaret Mc., aged thirty-seven years. Admitted February 23, 1910, complaining of edema of face, abdomen, extremities, and of pains in the back.

Present Illness. Two weeks ago she noticed puffiness about the eyes, most marked in the mornings. In a few days feet became swollen, and later her hands. The swelling increased each day, and finally the abdomen became involved in the edematous process.

Previous Medical History. Scarlet fever.

Examination. Slight edema of face. Heart enlarged to left; soft, low-pitched systolic murmur at apex. No ascites. Extremities edematous.

Urine: 1016; small amount of albumin; few fine granular and hyaline casts.

Blood-pressure: 125 to 100.

Result of treatment. Salt-free diet was used but not the Karell cure. On the 19th, owing to signs of impending uremia, a Karell diet with hot packs and caffein 0.2 gram t. i. d. were ordered, and the result was improvement in urine, chlorides, and in the patient's general condition.

Note. Chart shows absolute chloride retention despite increasing amount of urine. Note prompt and rapid elimination of chlorides and also marked diuresis on combining Karell cure (*i. e.*, Karell diet and medication).

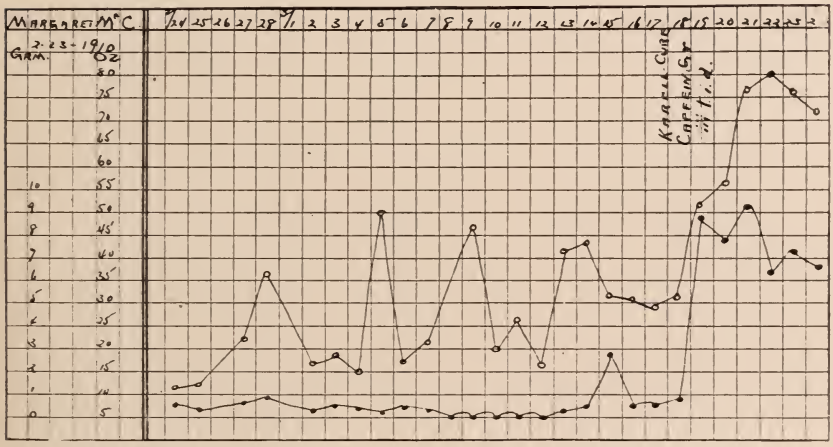

CHART XI

CASE XII.—Diagnosis: Nephritis.

Elizabeth W., aged thirty-six years. Admitted February 12, 1910, complaining of edema of extremities and abdomen.

Present Illness. Three weeks ago patient noticed slight swelling in the feet at night, the edema disappearing by morning, also puffiness about the eyes appeared at the same time. The swelling gradually increased, the entire body becoming edematous. During the summer of 1909 patient noticed some swelling in her feet which disappeared in a few days.

Examination. Anasarca. Heart not enlarged. Accentuation of both pulmonic and aortic second sounds. No retinitis.

Urine: 1013; large amounts of albumin; hyaline and granular casts.

Result of treatment. Rapid loss of weight, disappearance of edema. Patient discharged much improved.

Note. Chart illustrates the great loss in body weight, increase in urine, and marked chloride elimination.

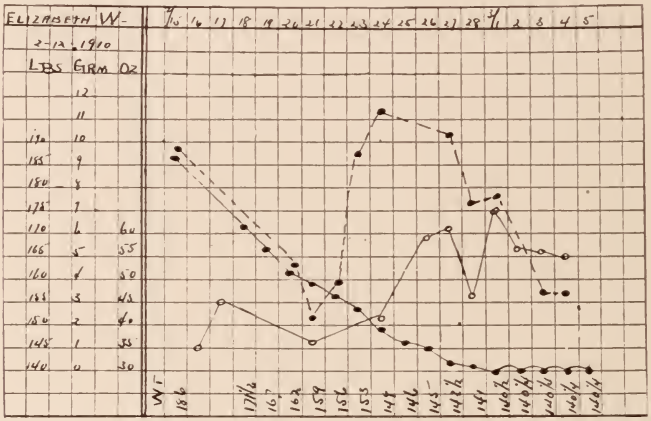

CHART XII

EFFECT OF THE CURE

1. *Subjective State.* The most important feature of the cure is that it gives the human organism the minimum of work. All strain on the heart is removed as far as is possible by the rest in bed, and by the very small amount of food given at frequent intervals, but furnishing enough nutrition under the circumstances, and providing fuel in such a form that it can be utilized with the least possible effort.

The good effects are seen by the patient almost before they are noted by the physician. The patient rests easier, there is a gradual cessation of subjective symptoms, headache, depression, vertigo, there is a return of the desire to partake of food where formerly nausea and vomiting held sway, there is an improvement in the respiratory condition, and the patient has less dyspnea, requires fewer pillows for his comfort, gradually assumes the prone

position, and is able to have long sleep without the intervention of morphin. These favorable changes are the reward of the strict observance of the Karell cure, and the strict observance consists in giving only 200 c.c. of milk at 8 and 12 A.M. and 4 and 8 P.M. The purpose of the treatment is immediately defeated if more fluid is permitted or if the patient is allowed to drink the milk whenever he pleases.

2. *Heart.* The effect on the heart is soon noticed apart from the subjective symptoms of restored compensation. Good effects are seen more constantly in those cases which are best termed myodegeneratio cordis, that type of cardiac disease seen in senility, emphysema, chronic alcoholism and in cachectic states (Wittich), and in 60 per cent. of this type of case improvement is seen. Valvular disease is improved in about 43 per cent., arteriosclerosis in 33 per cent., and according to Wittich, in nephritis, only 14 per cent., a percentage below that seen in my cases. Under the combination of rest in bed and the Karell diet the pulse assumes first a better quality and later becomes less irregular and less frequent. These effects are seen in many cases in which no medication is used, although we do not presume to deny for an instant the place digitalis preparations have earned for themselves in the treatment of arrhythmia and allorrhythmia. In some cases digitalis preparations must be used, but their action is materially reinforced by the use of the milk cure.

3. *Blood-pressure.* Blood-pressure is generally lowered, particularly in cases of renal hypertension, as may be seen from Chart X.

4. *Urine.* When the daily quantity of urine is closely watched it will be seen that there is marked increase in the amount. This, in some cases, begins within the first few days and reaches its height during the first week (Chart VII), in other cases, diuresis is delayed (Chart IV, Chart XII). Most authors state that the largest amounts are seen in the first two or three days, but this has not been the rule in all of my cases. At times there is a delayed diuresis, as has been described by His. The amount of urine passed in twenty-four hours may be two or three times the amount

of milk ingested, and as much as five liters has been observed. In the case of Mary S. (Case VII) almost 4500 c.c. were voided, and in this patient there was a loss of weight of 54 pounds in ten days. The specific gravity of the urine is variable, but, generally speaking, it is in inverse ratio to the amount of urine excreted. On the other hand, there has been noted a distinct rise in specific gravity without any change in the amount. The quantity of albumin diminishes and disappears when there is chronic passive congestion, and there is also a reduction in the number of casts; although this is not so apparent when a true nephritis is present. The effect on the phenolsulphonephthalein output is variable, but it cannot be said that there is any marked increase in the amount excreted.

5. The chloride output is generally very much increased over the intake. Repeated analyses by Hegler have given the percentage of salt in milk as 0.14, which would be about 1.1 grams in 800 c.c. of milk. By reference to Chart XII it may be seen what relation the output bears to the intake. Wittich regards this negative chloride balance as a good indication of the efficacy of the treatment, and attaches greater importance to it than to the twenty-four-hour quantity of urine in estimating the changes in the circulatory apparatus. Ordinarily there is a marked increase in the amount of sodium chloride during the first few days, both as to percentage and total amount. After two or three days the percentage falls, but the total amount increases with the increase in urine, and finally, as the urine amount returns to normal, the chloride balance is regained (Wittich).

To What is Improvement Due? An attempt has been made to ascribe the benefits following the Karell cure to three factors, namely, reduction in the amount of fluid, the minimum of salt contained in the milk, and finally to the melting of body protien. The last-named moment has probably some bearing on the effect of the cure when carried out in obese patients, but is scarcely to be considered as a factor in the cases of edema where as much as 31 pounds have been lost in three days (Mary S., Chart VII). Hence, in the cases studied as a basis for this paper, the so-called melting of body protein hardly calls for consideration.

Metabolic studies have been made by Hegler on patients suffering with renal or cardiac decompensation, or both, associated with obesity. He does not consider that the limited amount of fluid ingested alone accounts for the diuresis and loss of weight, and this he proves by a patient who took 800 c.c. of milk and a liter of infusion of lime-tree blossoms, and who nevertheless lost 10 kilograms weight in eight days.

To the low salt content of the diet has been ascribed much of the benefit resulting from the cure. During the first three or four days of the cure, patients excrete daily 8, 10, and 15 grams or more of NaCl; after five to eight days the excretion of salt sinks to 1 or 2 grams per diem, and when a diet containing from 1 to 3 grams of salt is given there is a transitory tendency to salt retention. If the NaCl equilibrium is followed for a longer period of time there is always a preponderance of salt excreted over that ingested (as much as 40 to 69 grams in three or four weeks). This is in contradistinction to Hedinger, who found a considerable salt retention and water retention, which, to his mind, accounted for the rapid gain in weight after completion of the cure (obese cases?). Hegler has made the interesting observation that salt added to the diet of an obesity case causes no water retention and no gain in weight, but when the same experiment is tried in dropsical individuals, there is an immediate cessation of loss in weight, with water and salt retention, and finally gain in weight. This latter experiment is by no means new, however, and was first performed by Javal and Widal. Somewhat similar results were also obtained by v. Hoesslin in normal individuals.

From this it would seem that the good effects might be ascribed to the salt poverty of the diet, but here again we find contradiction in a case described by Wittich.

CHART XIII

May 28, 1911	Salt-poor, without limiting fluid	700	3.0	4.2
May 29, 1911		700	3.0	3.5
May 30, 1911	Karell cure	1400	1.7	10.1
May 31, 1911	Karell cure	1300	1.7	9.2
June 1, 1911	Karell cure	800	1.7	4.4

This patient received for two days a salt-poor diet, but was allowed as much fluid as he desired. There was no increase in diuresis, however, but as soon as the Karell diet was given, although now but 800 c.c. of fluid was permitted, the amount of urine was doubled. A similar case is that of Case XI. It is difficult to see just how a reduction of salt intake (1.3 grams less than on the salt-poor diet) could account for the increase in urine and the remarkable amount increase in the amount of chloride.

It appears more probable that there are several factors which account for the efficiency of the Karell diet: (1) absolute rest in bed, which removes from the organism practically all work, and decreases the amount of work to be borne by the heart; (2) the low amount of fluid and food (800 c.c., containing only 27 grams protein, and furnishing about 520 calories) also limits the amount of cardiac effort; (3) the low amount of sodium chloride, (4) and finally, the effect of the Karell cure may be due to either the salt-poor diet or to the low amount of fluid, or it may be the sum of the two that brings about the brilliant results.

Hegler, as a result of his metabolic studies, decides that the value of the Karell cure cannot be judged from such studies, but must be estimated from the clinical side, and I would here call attention to Karell's remark quoted above. No matter to what factor or factors the good results are ascribed, the fact remains that in dropsical conditions of renal, cardiac and perhaps hepatic, origin, the Karell milk diet, given as taught by Karell, is the diet *par excellence*.

The Use of Drugs in Combination with the Karell Cure. In pursuing my studies the object has been to use only the Karell cure itself and to avoid, as far as possible, any medication. In many cases the milk diet alone brings about the desired improvement and no drugs are necessary, but in other cases medicines cannot be dispensed with. It has been noted frequently that patients who have been treated for a period of time with digitalis preparations without much improvement do particularly well on the Karell diet alone. On the other hand, cases presenting marked dyspnea, cyanosis, a frequent and irregular pulse, need relief more

rapidly given than is possible with the Karell cure by itself. Drugs such as camphor, digitalis, strophanthus, caffein, morphin, should be used in such emergencies; but it will be found unnecessary in the majority of cases to continue with their administration for any great length of time. Generally at the expiration of twenty-four to forty-eight hours there has been such a marked improvement that they may be discontinued.

When the Karell cure has been used for a few days, and when there has been no objective improvement, that is, when the quantity of urine becomes markedly decreased, when there is no change in the character of the pulse, and when the body weight and the edema remain as before, or when both increase, then drugs must be used. Of these, digitalis, theocin, caffein, and strophanthin seem to be especially suitable, and an observation of Jacob is worthy of repetition, namely, that whereas in these cases digitalis preparations alone, even in large doses, have had but little effect on the decompensated heart, when used with the Karell cure a much better result is seen, and much smaller doses of digitalis are required. This is especially to be seen when digitalis has been discontinued for a few days before beginning again with their administration.

Prognostic Significance of the Karell Cure. Jacob, Wittich, and others have called attention to the prognostic significance to the pure Karell cure, or the Karell cure used without medication. Wittich especially regards the prognosis as serious when there is chloride equilibrium, persistence of edema, and stationary urinary output or a diminution of the same, but particularly important is the chloride output by means of which one is enabled to decide on the prognosis by the first day. A positive chloride balance is absolutely unfavorable, and in his cases this was seen in but two cases, and these patients died within the first few days of the cure. Wittich also claims to see in the chloride metabolism not only an index of the extent of reëstablished compensation, but also of beginning compensation following a Karell cure. When at the end of a Karell cure there begins to be salt retention, he regards this as an early sign of a fresh decompen-

satory state—a preëdematous stage of cardiac decompensation, and he recommends a repetition of the Karell cure in order to spare the heart any further fatigue.

In 1910 I called attention to the unfavorable significance of this chloride retention, and a chart published at this time is reproduced here (Case XI). This patient was not on the Karell diet, but was given what is popularly called "a salt-free diet," but more properly a salt-poor diet.

Contra-indications. During our experiences with the Karell cure there have been no bad results when the treatment has been properly used. There are, however, cases which do not seem to respond to the diet, and for this reason the Karell treatment is no longer recommended in these instances. Patients exhibiting symptoms of uremia should not be put on the Karell cure, which restricts the fluid intake to a minimum, as it has been shown by Senator and others that in this crisis the fluid intake should be greatly augmented for the purpose of flooding from the system the unknown toxic substance or substances believed to be the the causative factor in uremia. Wittich states that the Karell cure in two cases of uremia left him absolutely in the lurch, and the patients were made materially worse. The treatment of such individuals should be that well known to all practitioners of medicine, and the Karell cure has no place whatever in the management of such cases.

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DISCUSSION

Dr. James Tyson: I do not know whether anyone here is old enough to remember, as I do, that some of Dr. Mitchell's early work, and perhaps the foundation of his little book on *Fat and Blood*, was the use of the Karell diet.[1]

Dr. James M. Anders: Karell's milk cure is much more used abroad than in America. I am quite sure that Dr. Goodman's paper is one of a very few written by American authors on the subject, and I feel that he deserves much credit for having given us such a complete presentation of the method, together with his own rather extensive personal experience. In the opinion of His, the Karell cure is not only effective in renal and cardiac dropsy, but also alleviates the disturbance in breathing and other distressing symptoms not dependent on the edema which many of these cases present.

Dr. Goodman referred to the fact that some authors recommended its use before actual decompensation takes place, and I am quite of the opinion that here is a very important field for the application of this method of dietetic treatment. For example, cases of aortic valvular disease, in which simply premonitory symptoms of failing compensation are present, often associated with a mild grade of arteriosclerosis, are those which respond most satisfactorily to this treatment. After positive involvement of the kidneys and the presence of uremia supervene it would be altogether contra-indicated. On the other hand, in these circumstances we should aid elimination by the use of large quantities of fluid taken internally. It seems to me that the Karell cure is after all a form of dry diet because of the very limited amount of fluid allowed. I have never been able to bring myself to the point of using it in valvular disease with dropsy in which the kidneys are intact. In all of these cases I feel that we ought to allow more food of a higher caloric value and aid elimination by other means, such as rest, cardiac stimulants, etc. In chronic nephritis, with or without dropsy, the treatment has been effective in my experience. Someone, I think Cheyne, has said that milk is blood. Recently, Kleinschmidt has pointed out that if to the milk we add a little iron a more complete food is the result; also that when milk alone was administered, there was a falling off in the hemoglobin

[1] Dr. Tyson now recalls a paper by himself on the "Milk Treatment of Disease" read before the Section on Practice of Medicine and Materia Medica and Therapeutics at the meeting of the American Medical Association, at its meeting in Washington, May, 1884, and published in the Journal of the American Medical Association, June, 1884.

percentage of the blood. It seems to me that this use of iron could be utilized to advantage with Karell's cure, particularly when the lengthened periods of its employment recommended by Dr. Goodman are followed. My habit has been, following the recommendations of His, to give 1000 c.c. for five or six days at intervals. I feel that this subject demands more thorough attention in this country than it has received in the past, and I earnestly hope that Dr. Goodman's paper will be the means of increasing interest in the subject.

Dr. Clifford B. Farr: One of the chief theoretical objections to this treatment is the fact that the diet is insufficient even for a patient in bed. I should think there would be no objection to adding sugar of milk or cream to this measured amount of milk. This would bring the nutritive value more nearly to the proper level and add nothing to the amount of fluid or chlorides.

Dr. Goodman (closing): I do not want to give the impression that patients are carried as long as the charts would indicate on milk alone. After three to six days we begin giving some food. During these days, however, the diet is limited to 800 c.c. of liquid in the form, preferably, of milk. The addition of cream or sugar of milk would probably be of advantage. I do not think, however, that a low caloric diet for three or four days or a week for a patient in bed and absolutely at rest can injure him to any great extent. I do not know how the cream or sugar of milk would be borne by a patient whose gastric mucous membrane is already congested as are the rest of his tissues by the failing powers of the heart. My patients have seemed to gain strength rather than lose it; probably due to the fact that they are in the hospital and absolutely at rest.

PRE- AND POSTNATAL WORK[1]

By H. R. M. LANDIS, M.D., THOMAS C. KELLY, M.D.
AND
CHARLES C. NORRIS, M.D.

The campaign having as its object the reduction of the infant mortality rate is undergoing the same evolution that has characterized other public health movements. In nearly every instance the initial step has been to treat individuals who have become ill. This unquestionably aids in saving the lives of many, no matter what the particular problem may be. But sooner or later it becomes apparent that the real solution lies in the prevention of disease. Those who have studied the causes of the high infant mortality rate have gradually come to see that marked progress is to be made only through the prevention of the illness common to young children rather than waiting until they become sick.

Except as it applies to the poor and ignorant, there is nothing new in prenatal or prematernity work. Women in the well-to-do class practically always consult a physician early in the course of their pregnancy and are under medical supervision until they have passed safely the puerperal period. Women of the poorer class, on the other hand, rarely consult a doctor until the time of their confinement is near at hand; and in many instances, especially among the foreign born, the services of a midwife at the time of confinement are all the attention the woman receives. Only too often inadequate care is the result of poverty. This is shown by our experience. You will get some idea of the social background

[1] Read April 5, 1916.

when you consider that in our series the size of the families ranged from two to eleven, and the income from nothing at all, or relief from a charitable agency, to from $3 to $20 per week.

The need of conserving infant life becomes apparent when it is appreciated that the very large proportion of deaths is due to preventable causes, and the solution of the problem lies in education. It is interesting to note that, as far back as 1876, W. L. Richardson[1] urged the necessity of instituting measures designed to reduce the morbidity and mortality rate among infants. His recommendations embraced practically every measure in force today; namely, frequent visits to the home, instruction in dietetics, day nurseries, playgrounds, maternity hospitals, etc. He ends his report with the following:

The study of public hygiene has already done much toward improving the sanitary conditions of our large cities and towns, but much remains to be done. The field for future work is large; unfortunately the laborers in it are few.

Of all the questions engaging the anxious thought of sanitarians and deserving the study of those interested in the welfare of the human race, there is none which should call for more consideration than that which deals with the problem how to save the lives of those helpless to help themselves. It is surely for the highest interest of the State to see to it that no means are left untried which may prove, in the least degree, of service in saving the lives of those who may be the very ones to whom, at some critical moment, the State may have to turn for its own salvation and defence.

Unless one is committed to the Malthusian theory, it must be admitted that a growing population is one of the surest indications of national strength. A low birth-rate or an unduly high mortality rate among infants is viewed with indifference or apprehension, according to the need for an increase in the population. It never has been shown that a nation is threatened with ruin which increases in size as the result of its own birth-rate; on the other hand, a gradually diminishing population means,

[1] From Boston City Board of Health Report, 1876: Report on Infant Mortality, by W. L. Richardson, M.D.

sooner or later, a weakened and inefficient nation. The need of conserving infant life is most apparent in sparsely settled communities where the development of the country is dependent on an adequate population. This is well illustrated in the Australian Federation. Here there awaits development a vast expanse of unoccupied territory and a relatively small population. For these reasons every effort is being made by the Australian Government to conserve infant life. Quite recently the government has made available a sum of money for every prospective mother unable to obtain proper medical care during and after confinement. In addition, nurses are being employed to supervise the babies during the first two years of life. It is furthermore proposed by the government to establish an adequate number of maternity hospitals for the care of women in the poorer classes.

Although this country is not at present threatened with a diminishing birth-rate, the need of prenatal and postnatal work may be urged on the ground of economy. It is cheaper to supervise infants and keep them healthy than it is to maintain hospitals for their care if they become ill. Looking at the problem in a broader way, it is one of the most effective means whereby sanitation and the laws of hygiene can be taught. The strongest appeal that can be made for cleanliness in and about the house is the effect this will have upon the child.

It is not necessary to recall in detail the various accidents which may occur during pregnancy and which can be averted if the prospective mother is receiving competent medical attention. And, while the need of such supervision is obvious, it is but a part of the greater problem having for its object the conservation of infant life.

Prenatal work has four objects in view: (1) the protection of the mother against the complications of pregnancy; (2) the improvement of the health of the mother that she may bear a healthy child; (3) arrangements for adequate confinement; and, most important, (4) the education of the mother as to the proper care of her baby after its birth.

Success in this phase of preventive medicine, as in every other

branch of public health work, is measured by the thoroughness with which it is done. The work, to be effective, must be intensive; failure is sure to result if the attempt is made to handle large numbers in a superficial manner.

The equipment necessary for the proper conduct of the work should consist: (1) of an obstetrician; (2) a nurse who has had training in obstetrics, social service, and municipal health work; (3) a pediatrist for the postnatal work; and (4) the services of an internist. The brunt of the work falls to the lot of the nurse, and care must be taken to see that she is not required to handle too large a number of cases. On her shoulders lie the responsibility of teaching the mother how to care for herself during pregnancy, and the more difficult problem of instilling into the prospective mother a knowledge of the laws of hygiene. The need of the services of an internist will vary. In the group which we report a considerable proportion (33.7 per cent.) of the women had either latent or active tuberculosis. It is obvious that team-work between the obstetrician and internist is essential in such cases, as it requires more careful supervision to maintain health, and, in addition, the question may arise at any time as to whether it is advisable to terminate the pregnancy.

It has been estimated that 75 per cent. of the maternal mortality and morbidity occurring as a result of childbirth is the result of preventable causes. In 1850, and even ten to fifteen years later, the mortality in many of the large maternity hospitals ranged between 10 and 15 per cent. from puerperal sepsis alone, which does not include the many deaths due directly to dystocia. Fortunately, since that period many advances have been made and the mortality and morbidity have been materially diminished. During the last ten years the maternal mortality in the Maternity Hospital in this city was 0.694 per cent. from all causes.

Undoubtedly great advances have been made during the last twenty-five years in the science of obstetrics, and as a result the mortality has gradually lessened. This is highly satisfactory; nevertheless, statistics regarding this point are very misleading. The difference in the results obtained in well-conducted maternity

hospitals and those secured from private practice in general are very striking, and, whereas the results from large hospitals are at least moderately satisfactory, those secured from general practice are far from what they should be. As most statistics are derived from institutions, where the better results are obtained, we are naturally more or less misled regarding the mortality from obstetric cases as a whole. Probably 85 per cent. of our women are delivered in their homes. The poor results often obtained in private practice are due to many causes, not the least of which is poverty. The proper conduction of a maternity case requires not only skill but time, and the fact that obstetric cases in general pay badly is a definite factor. Numerous visits by the obstetrician, the instruction in the hygiene of pregnancy and the hygiene of the puerperium and lactation, tests, measurements, etc., apart from the actual work of delivery, are time-consuming procedures and can hardly be expected for the small indemniture generally offered. Many other causes enter into this problem. However, the fact remains that obstetrics as generally practised outside of the maternity hospitals and that relatively small class of patients who can obtain skilful attendance is far from ideal. As a result the mortality and morbidity in the general run of cases are unusually high. This is especially so among the poor and ignorant.

Until at least very recent times even the best-conducted maternity hospitals in many instances did not do prematernity work. Often a patient with a bony pelvis so contracted as to preclude the possibility of a normal birth presents herself only at the onset of labor or, at best, such a short time before as to be of little value so far as the prophylaxis of dystocia is concerned. This is only one of many examples; malpositions, placenta previa, eclampsia, and many other conditions, the early treatment of which is of the utmost importance, are often allowed to advance, to the jeopardy of the woman and her expected child. Untreated tuberculosis, syphilis, and other diseases are also definite factors, increasing both the maternal and infant mortality and morbidity. Although these complications are not preventable, many are, and adequate treatment is of the utmost importance. These are well-

known facts, and detailed description would be unnecessarily time-consuming. In dealing with the class of cases treated at the Phipps Institute, the midwife enters largely into the problem. In this city efforts are now being made to improve these conditions, but this problem is such a large one and the questions so many-sided that advancement can only be made slowly. Cabot,[1] in an interesting article in a lay magazine, has recently emphasized the fact that the richest and poorest classes are the recipients of the best medical treatment.

The point which we wish to especially urge is that, while the well-to-do gravidara doubtless usually obtains adequate care, this is not so among the poor and ignorant, and even when the latter are confined in the best maternity hospitals their pre- and postnatal care is often far from satisfactory.

The need of better care is emphasized by Zinke.[2] He states that well-kept statistics, before this world war show that within the German Empire annually 8000 women die during labor and confinement. In this country, where statistics of deaths and diseases are poorly reported, it is conservatively estimated (by De Lee, of Chicago) that 20,000 women die every year of puerperal causes. In only one-fourth of these cases is death due to eclampsia, apoplexy, rupture of the uterus, hemorrhage, heart disease, or injury; in the other three-fourths the cause of death results from preventable puerperal complications. "Think what these figures mean. Accordingly, Germany has lost from puerperal causes during the last fifty years 400,000 women in the act of birth; in the United States of America, for the same period of time, 1,000,000 women have been the toll demanded by death in the confinement chamber alone. It may be that the figures are only approximately correct, inasmuch as our population has increased 40,000,000 during forty-five years. . . .

"One thing we cannot get away from is this: up to the present, in the world's history, more women have died from preventable

[1] Cabot, R. C.: American Magazine, April, 1916.

[2] Zinke, E. Gustav: Bulletin of the Lying-in Hospital of the City of New York, 1916, x, 303, 130–143.

cause in the confinement chamber than there were ever men killed in all the battles of the past and present. But this is not all; the morbidity of pregnancy, labor, and confinement is from three to four times higher than the mortality. These are appalling figures. They are undeniably correct."

It is with the hope of aiding the latter class of cases that the prenatal clinic at the Phipps Institute in Philadelphia has been instituted. The work at the Phipps Institute has been conducted along the following lines:

How Cases are Obtained. Our cases are referred to us for prenatal care by the Phipps Institute, Babies' Dispensary, and the Washington Avenue Dispensary. A few come to us from the Society for Organizing Charity and the Young Women's Union. A knowledge of the work is gradually spreading, and it is now not uncommon for women to apply on their own initiative, even early in the gestation. About one-third are tuberculous; the time of pregnancy averages about the fifth month.

Prenatal Care. At her first visit the nurse does the housing and takes the social history, thus getting the background of the case. She then goes over the prenatal instructions, advising patients as to the proper food and clothing, the necessity for exercise, general hygiene, etc. A specimen of urine is obtained and the blood-pressure taken. During the first few months of pregnancy the aim is to visit every two weeks; the eighth month, every ten days; and the last month, every week. The blood-pressure is taken every month and a specimen of urine secured at each visit; if any abnormality is present it is reported to the physician in charge and the patient told to come in for treatment. The proper kinds of baby clothes are advised, and patterns are furnished if the patient is able to use them. As many of our cases are receiving help from some charitable organization, and none of them have adequate incomes, this is not often done. We are fortunate enough to have layettes furnished for some of our more needy mothers, particularly for the Jewish ones.

Confinement. In some instances they can afford a private doctor or have a family physician who will attend them, but if

they have no one and prefer remaining at home they are given a note to one of the maternity dispensaries. Those who previously have had midwives are advised to either engage a physician or to arrange for confinement in a maternity hospital. Those patients who prefer a woman physician are referred to the Woman's Medical College, Maternity Department. When the patient remains at home, instructions are given as to the supplies needed. If a hospital or maternity is advised by the physician, or the patient suggests such a course, she is given a note to one of them, and we see that a bed is reserved and arrangements made for her confinement. If there are children, we see that they are suitably placed, referring them to the Children's Bureau.

Postnatal Care. After confinement no visits are made, so long as the visiting nurse is in attendance, which is usually for eight to ten days. After she leaves, a call is made and instructions given as to the bathing, feeding, and general care of the baby. The patient is urged to come to the Gynecological Clinic as soon as possible for postnatal examination, and, if tuberculous, for a chest examination as well. As soon as the baby is three or four weeks old the mother is asked to bring it to the Prophylactic Dispensary of the Babies' Hospital, where it is weighed, the temperature taken, and advice for keeping it in health given by the physician, who requests that it be brought every two or three weeks for observation. If it should be ill, it is referred to the Dispensary for Sick Babies of the Babies' Hospital. This closes it as a prenatal case, and it is carried as a postnatal one, a visit being made each month to see that the instructions are being followed. If the child is not brought to the dispensary after three months (a visit being made every month and the necessity for keeping it well, instead of waiting for it to become ill before taking it to a physician being emphasized) the case is completed.

Our total number of cases to March 1 was 175. Of these, 61, or 33.7 per cent., were tuberculous. There were but five maternal deaths in the entire series. One patient, non-tuberculous, died of malignant endocarditis; another, also non-tuberculous, died from edema of the lungs and dilatation of the right heart, following a

Cesarean section. Three advanced tuberculous patients were treated in Phipps Institute until the time of delivery, when they were removed to a maternity hospital. They lived some weeks after confinement, as did their babies. The time in which our cases come to us varies from the second to the eighth month, the average being the fifth month. They have been cared for as follows: not yet delivered, 23; closed because of removal from district or because they were uncoöperative, 26; not prenatal, 3; unattended, 2; in general hospital, 22; in maternity hospital, 16; by dispensary doctors, 56; by private doctors, 15; by doctor and midwife, 1; by midwives, 11. Total, 175.

Of five miscarriages, two of the women were tuberculous; of four stillbirths, one of the women was tuberculous; and of eleven infant deaths, eight were children of tuberculous mothers.

The number of women who manifest evidences of active pulmonary or laryngeal tuberculosis, either during pregnancy or during the puerperal period, is sufficiently large to command attention. In some instances it is known that such a condition exists, but in a far greater number serious damage has resulted before the attending physician realizes the danger. The prevailing opinion is that the effect of pregnancy on a tuberculous lesion is most serious, and in a measure this is true. Most of the statistics dealing with this question, however, have been furnished by obstetricians, who are often consulted after the pulmonary damage has become irreparable. Many unfavorable terminations could have been avoided had the attending physician taken an adequate medical history. Thus, if the woman gives a history of having had a member of her immediate family die of tuberculosis, or if she herself has ever had an attack of pleurisy, a hemoptysis, or a persistent cough, attention should at once be directed to the conditions of her lungs. The experience at the Phipps Institute has shown very clearly that forewarned is forearmed, and that coöperation between the medical men and the obstetrician will carry many of those women safely through their pregnancy.

Fifty cases of pregnancy in tuberculous women have been thoroughly followed in the Prenatal Clinic of the Phipps Insti-

tute during the last two years. These cases recently have been reported in detail by one of the authors.[1] In this series there was a morbidity of 22 per cent., in contradistinction to the 60 to 80 per cent. morbidity usually observed. There was an infant mortality of about 11 per cent. in the place of the 50 to 60 per cent. mortality generally noted. Many authorities view the incident of pregnancy in a tuberculous woman as an indication for emptying the uterus, regardless of the pulmonary condition and the advancement of the pregnancy. This tendency is especially present among the Germans. We feel that such a dictum is far too sweeping, and believe that this stand is confirmed by our results. It is not to be understood that we do not believe in therapeutic abortion in certain cases. This has been practised in five of the fifty cases above referred to. Intervention, to be effective, should be employed prior to the fifth month, and should be done early; that is, immediately upon the onset of any acute symptoms. Such a plan exposes the patient to the obvious risk that she may do well for the first half of pregnancy and suffer an exacerbation during the latter months.

It is impossible to escape the well-established fact that every tuberculous woman who becomes pregnant is exposed to a distinctly added risk thereby. Doubtless a better plan in the majority of cases would be for the woman to avoid conception, and such advice on the part of the physician is undoubtedly the safest to give. In exceptional cases, where the disease is limited and nonactive and has been in abeyance for a period not less than two years, and when the patient is willing and able to obtain adequate treatment, and a child is especially desirable, it is probably justifiable to countenance conception. Under all circumstances the patient should be under the care of a skilled internist and should receive regular and frequent examinations.

All our tuberculous cases have been kept under close observation and the necessity for treatment emphasized to them. In this connection it should be remembered that tuberculosis is only in

[1] Norris, Charles C.: Pregnancy in the Tuberculous, Amer. Jour. Obstetrics, New York, April, 1916.

the rarest instance congenital, the infection in these infants being postnatal in practically all cases. For this reason, and for the maintenance of their own health, these women should not nurse their infants, and the children should be especially guarded against postnatal infection. It would be highly desirable to have a Wassermann test made on each woman when she first comes under observation. Up to the present time it has been impossible to have this done, but in the near future we hope to include this in our routine work.

While the report on the prenatal work is based on a study of 175 cases, the postnatal care includes results of only 100 babies, as the Prophylactic Dispensary was started some time after the prenatal work at the Phipps Institute had been in operation.

The report of the postnatal work in the prophylactic care of babies is based on a study of 100 cases which have been under our care in the Prophylactic Dispensary of the Babies' Hospital and Phipps Institute. The mothers of fifty-two of these patients received prenatal care and were referred from the Phipps Institute; the remaining forty-eight were referred direct to the dispensary from various sources for observation in the Prophylactic Dispensary. In considering the statistics of the cases it seems preferable to consider the total number together, merely contrasting those from different sources for purpose of comparison as to the final results.

Of the 100 cases upon which this report is based, 36 are closed cases—cases which paid only one or two visits to the dispensary and then for various reasons were no longer under observation, and 64 active cases in which we still have more or less constant supervision.

As most of our cases are of necessity from the surrounding territory, the situation of the dispensary would naturally have quite some influence on the nationality of the patients: thus 46 per cent. of the total number of cases are of Hebrew extraction, 29 per cent. of Latin extraction, and the remaining small number divided among the Slav, negro, Teuton, and Celt. In 29 per cent. both parents were born in Russia, in 19 per cent. in America,

in 18 per cent. in Italy, and—a rather interesting fact—in twenty-two instances the fathers were native-born Americans, but the mothers from foreign countries, principally Russia and Italy.

In considering the family history only one fact could with any positiveness be ascertained; namely, the presence or absence of tuberculosis. As regards the other diseases, notably syphilis and alcoholism, very little of a definite nature could be extracted, although in three instances there were quite visible evidences of alcoholism in the father, and in one case a positive Wassermann reaction was obtained from the mother. Out of the 52 cases referred from the Phipps Institute there was a history of tuberculosis in one or more members of the family in 24 cases, nearly 50 per cent., the mother suffering in all except two instances, or in 22 out of the 52 cases. Of the 48 cases referred from outside sources, 8 showed a family history of tuberculosis, the mother suffering with others in 4 cases.

As to the closed cases, of which there are 36 out of the total number of 100, they were considered closed either because they had not been to the dispensary for a period of one year, or else, because of some good reason, they had ceased to be visited by the nurse. Of these, twenty-one, or about 58 per cent., paid only one visit and were never seen again, and only twelve of the thirty-six had been seen at all by a doctor; most of them went to the dispensary before it was in full running order. They ceased their visits for various reasons: most of them said it was too far to come to the dispensary, several moved out of town, others were indifferent to the advantages to their children by such care, and still others came for mere pecuniary advantages, and ceased their visits when it was found that free milk was not forthcoming on their first visit.

A study of the health of the cases still under our care is rather hard to particularize in statistics, but, for purposes of comparison of the cases with tuberculous history and those without, the gain per baby was divided by the length of time under observation, and the following deducted: The cases of tuberculous families made an average gain of $16\frac{7}{8}$ pounds for twenty-two and three-quarter months, or about $\frac{3}{4}$ of a pound per month, and the non-

tuberculous cases made an average gain of $10\frac{1}{5}$ pounds for nine and two-third months, or a little over a pound per month—a very satisfactory gain when all the circumstances are taken into consideration.

As to the care of the patients themselves: Most of our cases are told to come back in usually from two weeks to a month, depending a great deal on the particular need of the individual, and the nurse makes her visits to the house usually in about the same averages —at least once a month in all cases, and oftener if found desirable, even going several times a week in certain particular instances.

The feeding of the patients has been as simple as possible. The great predominating fault with the breast-fed babies is that they are nursed too often and too irregularly, and the cases least amenable to improvement are the ones which it is impossible to convince the mother that the child should be nursed at regular hours, and not when she thinks of it during the daytime and when the child thinks of it at night. Wherever possible, breast feeding has been persisted in as long as practicable, and only as a last resort has artificial food been advised in the very young infants, except in cases of mothers with tuberculosis, when they had been taken off the breast immediately. Very often we have been able, by increasing the food supply of the mother, to increase her own milk and prolong the nursing period, and have shown in many cases an appreciable gain in weight in the infant. When nursing was no longer possible, artificial feeding was commenced, and then our problem became more complex, as it was not so much what the infant could have to eat as what they could get to eat.

In the modification of milk only the simplest dilutions were in most cases advised, even no attempt at top-milk formulas being attempted; in most cases it was hard enough to get the mothers to understand what half-milk and whole water meant. Milk with water or barley-water as a diluent and the addition of sugar was usually all that was given; at times the sugar even was omitted, as the parents were unaccustomed to such luxuries. The mother, in the presence of the nurse, was told what to give, and then the following day visited by the nurse and instructed in the method

of its preparation. For some time milk, bottles, and nipples have been sold at cost to the patients. Lately we have been able to get started a small milk fund, whereby we have had delivered to the homes of the deserving a quart of good milk—for the mother in case it was advisable to try and increase her breast supply; for the baby if it was on artificial feeding. This has been a wonderful help, and in several instances has been the turning-point in the health of the baby.

In general our results have been quite satisfactory. Out of the entire list of 100 cases during the part year we have had only one death—a case which was seen only once, never returned, and died some time during the past summer from some, to us, unknown cause. Also, except for several minor and temporary ailments which have arisen from time to time, we have had only three sick children—two during the past summer from severe enteritis, and one who just recently became ill—all three strangely in tuberculous families. Except for these instances, the infants have all done remarkably well when one considers the disadvantages under which they labor—ignorance, poverty, and filth. As a whole, the mothers have proved quite receptive and anxious to do their share in the promotion of their babies' health. This has been due in great measure to the kindness, wisdom, and tact of Miss Roche, our visiting nurse; for, while it is true that the parents wish to see a doctor and hear from his lips the direction of the baby, yet it is equally true in such work that without the services of a well-trained and capable nurse the directions would lose half of their value.

We have been greatly aided by the effective coöperation of the University Hospital, Woman's Hospital and Barton Dispensary, Maternity Hospital, Jewish Maternity, Jefferson Maternity, Southeastern Dispensary, and Polyclinic Hospital.

The work has been made possible by the very effective manner in which it was organized by Miss Stringer, head of the Social Service Department of the Phipps Institute.

DISCUSSION.

Dr. Samuel McC. Hamill: I do not know exactly when the Phipps Institute began its intensive work in prenatal care, but there has been intensive work in this same line carried on by the Health Centre of the Child Federation for almost two years. The Centre is located in the Italian district, and its work is limited to fifteen city blocks. The purpose is to educate all the people in these blocks in the methods of maintaining their health. As a part of the procedure we take care of all the pregnant women living in the district. The work of the Centre has been carried out in the following fashion: We started in the most eastern and thickly populated block in the city. Having obtained a house to house census of this block, and having studied its sanitary condition, our workers visited each home to teach the people living in it how to keep themselves well. All the pregnant women in the block were located and so far as possible were brought by the workers to the Centre building for examination. The result of our efforts interested us very much. At first only about 20 per cent. of the women came to the Centre for general and local examination by a physician; now, about 50 per cent. come voluntarily. Those who do not come are kept under observation, through regular home visits, by the nurse. Urinalyses and blood-pressure readings are regularly recorded. We have had up to the present time 219 cases delivered. We have had no deaths among the mothers. We have had 4 stillbirths; in 3 of these the mothers were delivered by physicians and in 1 by a midwife. There have been 2 miscarriages prior to the sixth week. This manner of procedure has two striking advantages over all other plans for conducting prenatal work: one is that every pregnant woman in the district is looked after, and the other that the women are under observation almost from the beginning of pregnancy. The majority of our cases come to us prior to the third month. We have had 4 deaths among the babies, 2 of congenital syphilis, 1 a few minutes after birth; and twins born to a syphilitic mother died within an hour or two. We have under observation at present 64 pregnant women. We have not been able to follow all cases that have come under our care to their termination, because some of them have moved out of the district before term. Some of these we have followed by correspondence and through other agencies. I am sorry that I cannot give a more detailed account of the work, as our method of procedure and results are of special interest.

Dr. William R. Nicholson: I am extremely interested in this paper. It undoubtedly is a sign of the times, "prenatal and postnatal care." Some of our friends do not approve of it. I have had a very

well-known physician say very recently that it is very much better to keep hands off in order that the physically unfit might perish, that it is better for the race. A great many of us do not believe that, hence the prenatal and postnatal dispensary. Under the requirements of the State Bureau of Medical Education every hospital must give its internes a certain number of obstetrical cases. Therefore we at the Polyclinic established an out-patient service and a prenatal clinic. We had previous to that time done the thing in a sort of half-hearted way, but for the last year or year and a half we have been carrying on this work routinely. The pelvic measurements are taken, the urine is examined regularly as is also the blood-pressure and the Wassermann and gonorrheal fixation tests are also made. Undoubtedly, as Dr. Landis said, the employment of the Wassermann test will be one of the most important factors, with the exception of the examination for tuberculosis.

I feel compelled to say a word about the class of workers known as midwives. Dr. Landis took the attitude, which I rather think is that of the profession generally, of antagonism. I do not think the profession realizes the exact situation concerning the midwives. We have about 200 in this city who are registered with the State Bureau—I have the honor of representing the Bureau in that work. We have about 200 women who hold our certificate. We have in the twenty-one months ending last October 12,977 cases delivered in this town under our care. These reports are all made to me. Of that number, all but 10,428 were inspected after delivery by graduate women physicians in the employ of the State. In the event of any complication those patients had the benefit of observation by the inspectors, or the physician of their own choosing, or they were sent to hospitals and delivered by a member of the hospital staff. We had a fetal mortality of 365 cases in the first ten days of life, including premature cases; the maternal mortality was 20. I wish to emphasize that when you gentlemen advise a woman, coming to a prenatal dispensary, not to go to a midwife, but to a doctor, you are not carrying out your full duty, in my humble opinion. If I were a woman and going to give birth to a child and had any assurance that there would be a normal delivery, I would rather have any one of fifteen or twenty midwives that I know take care of me than be turned loose upon the hands of the ordinary "garden variety" of practitioners in this town. If you are going to do away altogether with the midwives you must supply doctors qualified to do the work. The midwife is a necessity and she is going to stay as long as any man in this room is alive. The men who advise against the midwife, but provide no substitute, take the same attitude as Dr. De Lee. He believes that the evils of the midwife system are so great that he is willing to shut his eyes to the present loss of life

among the women and babies because of the future good which in his opinion will result from the abolition of the midwife. Such men are perfectly willing to allow women to die of gonorrhea, syphilis, or sepsis, or from neglect. The strongest argument against the midwife, as I see it, is not in the delivery and postdelivery care, if done under police surveillance such as we exercise over them, but that we cannot exercise prenatal care in their cases. We have no way of controlling prenatal care among midwives. If our work could be extended to include this department it would be of great value.

Dr. Charles A. Fife: The authors of the paper are to be congratulated upon their report of such prenatal work and also upon the establishment of the first dispensary of this kind in Philadelphia. The work is the most profitable and promising means of lowering infant mortality. One-third of the total death-rate of babies occurs in the first month due to prenatal cause. By such work not only is this early mortality lessened, but also the mortality of later months is lowered by lessening the likelihood of diarrheal and respiratory diseases because of the better care, the improved hygiene, sanitation, etc., resulting. Reports show that children born of mothers who have been watched during pregnancy weigh from 4 to 8 ounces more than babies born of mothers who have not been watched, carefully guarded, and properly fed during pregnancy. This means that the former class of babies have much more vigor and have much better resistance. Then, too, the instruction given by the nurses upon hygiene and sanitation lessens the ravages of diarrhea. Their work in encouraging mothers to nurse their babies is the biggest factor in prenatal care.

Dr. William N. Bradley: I have been much interested in this subject and have done some work along these lines. At the Starr Centre during the year 1914–1915 we had under our care 83 expectant mothers practically as outlined by Dr. Landis. At each visit of the nurse the urine was examined and the blood-pressure taken. The average period of care was four and a half months; the average number of visits, nine and a half. Of the 61 carried to the end of pregnancy, 8 were delivered by midwives and 38 by dispensary physicians; the remainder by private physicians. There was no case of abortion. There was 1 premature birth, 1 difficult labor with living fetus, 1 stillbirth; 59 babies were living and well at the end of the first month of life, and 58 of these were being entirely breast-fed and the remaining one partially breast-fed. The paper and discussion are important in showing the value of the work being done down town among this class of people.

A NEW "MUSCLE SUBSTITUTION" OPERATION FOR CONGENITAL PALPEBRAL PTOSIS[1]

By JOHN B. ROBERTS, M.D.

(A Contribution from the Surgical Laboratory of the Philadelphia Polyclinic)

In November, 1914, a three-year-old girl was sent to me by Dr. H. A. Stout, of Wenonah, N. J., for operative treatment of very marked double ptosis and a moderate degree of epicanthus of both eyes. The child had been taken previously to an ophthalmic hospital, but no operation had been performed. She had

Fig. 1.—Arabella S., at three years of age; double congenital ptosis and slight epicanthus.

become very shy and sensitive because of the facial deformity, and could not see very far in front of her unless she threw her head back to get vision through the narrow slit between the eyelids. She would not look at me or anybody else, but hid her face with her hands when one attempted to examine her.

My experience with operations for ptosis, admitted to be limited, has never been entirely satisfactory. Reading also has led

[1] Read April 5, 1916.

me to believe that the experience of active ophthalmic operations has been often similarly unsatisfactory. I devised, therefore, an operation which I had suggested in 1912,[1] based on the myoplastic method, which we surgeons use in traumatic and orthopedic

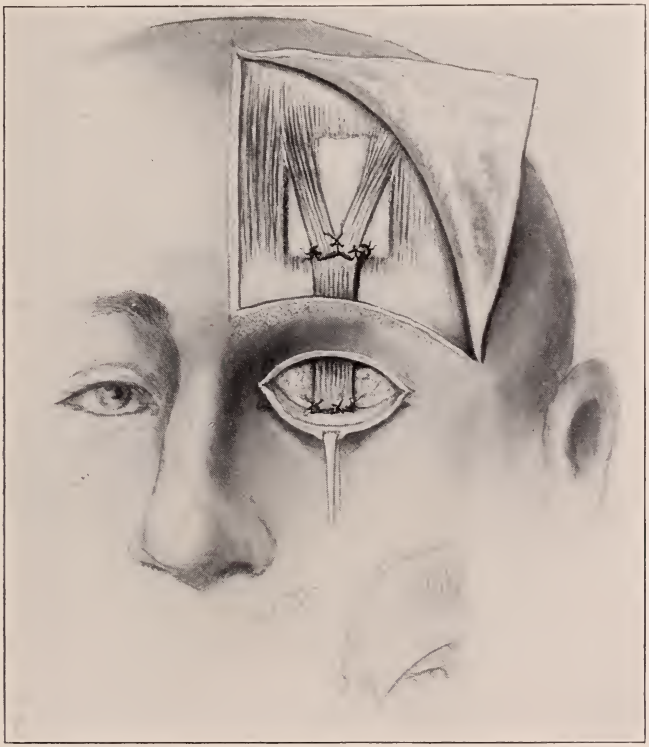

FIG. 2.—Diagram of Roberts's "muscle substitution" operation for congenital ptosis by transferring fibers of occipitofrontalis. Operation performed March 29, 1916, on Arabella S. Small diagram shows method if flap is turned downward and outward instead of upward and outward.

muscular deficiencies. It seemed, however, best to try at first one of the already recognized ophthalmic procedures. I, accordingly, operated on both eyes by the Wilder method.

[1] Surgery of Deformities of the Face, Wm. Wood & Co., New York, p. 221.

The child could uncover the eyeball after this operation, so that in the right eye about one-half the cornea was visible. The left eye was not so satisfactory in result, though the lid could be lifted somewhat better than before operation. The result of the operative treatment employed was encouraging, however, to the parents; and this year (1916), in March, the little girl, being now about four years of age, was brought back to me for further treatment. At the time of the first operation I had removed a vertical ellipse of skin from the front of the nasal bones to lessen the epicanthus, which deformed both eyes to a limited extent. Not much was gained by this operation, however.

FIG. 3.—Arabella S., aged four years. Photograph shows effect on left eye of frontalis "muscle substitution" for congenital ptosis one month after operation.

I determined upon the present occasion to lessen the epicanthus by an arrowhead-shape excision of skin and superficial fascia by Berger's method, to get rid of the deformity at the inner canthus of the eye; and to try my previously devised muscle substitution method, which I had employed only in the cadaver, though I had suggested it four years before, to relieve the ptosis of the left upper eyelid. The left eyebrow was shaved, and an incision carried from the root of the nose along the superciliary ridge almost to the external angle of the frontal bone. From the

nasal extremity of this cut a vertical incision was made through the tissues of the forehead almost to the hair line. The flap was turned upward and outward so as to expose the occipitofrontal muscle and tendon. Just beneath the upper orbital margin an incision down to the fascia of the upper lid was made from the nasal to the temporal side, following the curve of the bone. The skin flap was turned downward and the tarsal plate exposed and its upper edge indentified. A tunnel was then cut beneath the soft tissues, about half an inch in width, extending under the orbicular muscle to the incision made through the shaved eyebrow.

FIG. 4.—Dr. Roberts's second case before operation. John K., aged eight years. Double congenital ptosis, with congenital palsy also of the recti except the interni. Left eye was operated upon by same method as the case in Figs. 1 and 3. There were almost no muscle fibers uncovered in the frontal portion of the occipitofrontalis, and result was not satisfactory. If a second operation is done, the fibers will be brought up under the hair of the scalp.

From the muscular belly of the occipitofrontal muscle, immediately above the tunnel opening, was cut a strip of muscular fibers about a third of an inch wide and an inch and a quarter long. The parallel incisions making this strip diverged a little at their upper ends so as to make the muscular band wider near its upper extremity. A cross-incision was made at the upper end, converting the strip into a long flap.

This flap was turned downward, thrust through the tunnel, and attached to and upon the upper edge of the tarsal plate by three silk sutures. The two corner sutures were put in as mattress stitches and held the flap on top of, that is, superficial to, the

tarsal fibrous plate. Returning to the frontal region, I cut on each side of the turned-down flap two strips of muscle, each half the width of the inverted flap, leaving them attached to the muscle above. These were drawn toward the fold of the inverted flap, attached to it on its superficial surface, which formerly had been the under surface, by a mattress suture at each edge, and were united in the middle line by a third suture, also put through the turned-over portion of flap so as to make a mattress suture. An additional suture was inserted at one edge where the first flap was bent over to keep it thus folded. The skin and superficial flap of the forehead were then replaced and sutured in position by one worm-gut suture. (The result at present is excellent, though the time has been only four weeks.) Subsequently, the Berger operation, with arrowhead excision on each side, was performed for the epicanthus. The sutures were so placed as to lift a little the canthus of each eye. The wounds all healed by first intention. A satisfactory fact, in addition to the ability to raise the eyelid, has been that the operation makes a normal furrow in the lid at the seat of the upper edge of the tarsal plate. This is conspicuously absent in cases of congenital ptosis.

If the incision through the eyebrow to that at the middle line of the forehead for any reason seems undesirable, the flap can be turned outward and downward instead of outward and upward by making the horizontal incision within the hair line instead of in the eyebrow. It probably makes very little difference which incision is made unless the superciliary one employed in this instance should interfere too much with the branches of the supra-orbital nerves and vessels. These will be less disturbed if the horizontal cut is made at the top of the forehead through the hair instead of at the bottom through the eyebrow.

When the *muscle* fibers of the frontalis are poorly developed in the forehead region, it may be necessary to carry the sagittal incision far upward into the scalp, and thus uncover muscular tissue beneath the scalp itself. I should be willing to go even beyond the coronal suture to obtain a thick mass of muscle for the three muscular bands. Transferring tendon alone in this operation will probably be futile in result.

MENTAL DISEASE AND MENTAL DEFECT, THEIR MAGNITUDE AND IMPORT[1]

By OWEN COPP, M.D.

It is a common fallacy of the public mind, and especially of the mind of the legislator, that mental disease and mental defect may be neglected without serious consequences; that the burden of such afflicted may be evaded by inaction, and, therefore, that deficient public expenditure and effort in their prevention and treatment may be tolerated in the interest of economy. This fallacy is grounded, not in parsimony nor want of humanity, but in deep unconsciousness of the magnitude and import of the matter, and yet it is of universal concern to the individual, as well as the State, in every station of life, in every family high or low, wise or ignorant, rich or poor.

Mental disease and mental defect are multiform, but, in the gross, affect three classes of individuals, the insane, the feeble-minded and the epileptic. These are closely allied in the nature of their needs and in social and economic significance. They are about equal in prevalence. Together they present different aspects of one great problem.

The number of insane enumerated in the last United States census, 1910, is 187,791 in 366 institutions, an increase in six years of 37,640, or 25.1 per cent. The number of insane admitted to these institutions in a single year, 1910, was 60,769, an increase over the sixth year previous of 11,147, or 22.5 per cent. During the same period the general population of the United

[1] Read May 3, 1916.

States increased about 12 per cent. These percentages, however, do not necessarily signify a relative increase in the prevalence of insanity in the community, but they prove beyond question that the public burden is increasing by accumulation of insane in institutions more than twice as fast as the general population.

Furthermore, these figures express only the average for the whole country, whereas a wide variation obtains in different States, according to age, density of population, completeness of enumeration of the insane, sufficiency of provision for their care, liberality of laws encouraging their treatment in hospitals, extent of immigration, and other causes. For example, Pennsylvania falls somewhat short of this average, having in 1910 but 196.4 insane in institutions per 100,000 of her general population against the average 204.2 while her admission rate was 58.7 against the average 66.1. In marked contrast the adjacent State of New York had on the same date 343.2 insane in institutions per 100,000 of her general population, a ratio 74 per cent. larger than Pennsylvania's, while her admission rate was 93.6, exceeding Pennsylvania's by 59 per cent. In even greater contrast are such percentages in Massachusetts, which exceed those in Pennsylvania by 75 and 114 per cent. respectively. The older countries, Scotland and England, are in close accord with New York and Massachusetts. Obviously, then, the actual burden of insanity is far greater than appears in the United States census.

The deficiency of enumeration and of provision for the feeble-minded and epileptic is universal and extreme, far greater than such deficiency for the insane. The last United States census states that a conservative estimate of the number of feeble-minded then in institutions and the community would be 200,000, whereas only 20,791, about 1 in 10, were enumerated under care in institutions. Notwithstanding the increasing interest and activity in extension of provision for this class there were still seven States in the South and West which did not provide for them outside of almshouses and penal institutions. Pennsylvania compares favorably with other States in such provision, but every State lags far behind in meeting the reasonable demand.

The epileptic are, probably, as numerous as the feeble-minded, but their plight is even more deplorable.

The annual cost of the burden of insanity alone, computed at the conservative estimate of $175 per capita of insane in institutions in 1910, would amount to $32,863,425 in all the States of the Union. New York expended in 1914 in the care of about one-sixth of such insane, $7,225,015. Massachusetts spent the same year in the care of the insane, feeble-minded and epileptic in her institutions $5,109,111, which was 27 per cent. of the whole cost of her State government.

The growth of this burden is so rapid and persistent as to present one of the great problems of state finance.

Nevertheless, this visible presentation is relatively insignificant in comparison with the reality, the mere pinnacle, as it were, of a social iceberg whose bulk is buried in the obscure depths under the surface.

It does not show the cost (exceeding the rate in hospitals) of home care and treatment of the insane and mentally affected in the community, nor the loss of income from productive labor of these and others in institutions, nor the handicap of the wage-earner and bread-winner by a dependent in the family, nor the consequences of stress and overstrain upon relatives with similar inheritance predisposing to mental break-down, all together constituting an indirect burden as great, probably greater, than the direct burden.

It ignores the collateral burden of inefficiency, vagrancy and pauperism, of delinquency and crime, of alcohol and drug inebriety, of sex immorality, illegitimacy, prostitution and spread of syphilis and other evils whose causation is assigned by the best expert opinion to mental defect and mental disease as large factors.

Moreover, these are but the tolls exacted of the passing generation. What of the future? Read in answer Goddard's tragic story of Martin Kallikak and his descendants through six generations in two distinct lines of direct descent; one from sound stock through his lawful wife, the other through the feeble-minded daughter of an inn-keeper.

The 496 descendants from sound stock uniformly in each generation represented the best citizenship in their communities and had among their number no feeble-minded, only one insane, no epileptic, no illegitimate children, no immoral women, one male sexual degenerate, no keepers of houses of prostitution. The appetite for alcoholics was prevalent among them at a period when it was common everywhere, but it made only two habitual drunkards. Fifteen children, only, died in infancy.

On the other hand, the 480 descendents from the feeble-minded strain were allied in each generation always with the inefficient and degraded in their communities and had in their number 143 feeble-minded, three epileptics, three criminals, 36 illegitimate children, 33 immoral persons, mostly prostitutes, eight keepers of houses of prostitution, 24 habitual drunkards, while 82 died in infancy. Only 46 descendants, or 24 per cent. of known cases, were considered normal individuals. This remarkable example may exaggerate the danger of hereditary taint, but it suggests possibilities of grave import. The menace to the future is the more threatening because the partial corrective has been removed by the altruism of the present, which protects the weakling, who otherwise in early days succumbed to a hostile environment.

The actualities of the present situation and the possibilities of further harm force the conviction that the burden of mental disease and mental defect cannot be evaded by indifference, that the load grows heavier with inaction, and the only avenue of escape leads to militant attack upon the whole problem.

The first step in advance invades the field of investigation for clear definition of the problem itself and its exact relations to social and economic conditions and movements. Encouraging progress, though far short of the requirement, has already been made by various public and private agencies, notably the following:

1. In knowledge of the pathology, causation, diagnosis, and treatment of mental disease through clinical observation and laboratory research in hospitals for the insane, particularly the McLean Hospital, Massachusetts, the Michigan Psychopathic Hospital, Ann Arbor, the Boston State Psychopathic Hospital,

and the Phipps Psychiatric Clinic of Johns Hopkins University; also in knowledge of the morbid anatomy of the defective brain by the studies of Drs. Walter E. Fernald and E. E. Southard at the School for Feeble-minded, Waverly, Mass.

2. Progress in support of the theory of heredity and the importance of eugenics by extensive field work and investigation, conspicuously by H. H. Goddard in the psychological laboratory at the Vineland Training School and by C. B. Davenport in the Eugenics laboratory at Cold Springs Harbor, New York.

3. Progress in the acquirement and dissemination of information concerning existing conditions, in extension of provision and betterment of methods of care, of the insane and mentally defective through surveys, publications, lectures, mental clinics, conferences, and exhibits, by or under supervision of the National Committee for Mental Hygiene, its State societies and allied associations, the Rockefeller Foundation, and philanthropic individuals; by the extension department of the Vineland Training School originally, and its successor, the Committee on Provision for the Feeble-minded; and by special reports of public boards and commissions.

4. Progress in establishing the true relation of mental defect and mental diseases to delinquency and crime by special examination of the mental condition of inmates of reformatories and prisons and the findings of expert investigators and advisors of juvenile courts, especially of Dr. Wm. Healy, director of the Psychopathic Institute of the Chicago Juvenile Court.

5. Progress in revealing the remarkable prevalence of mental defect among children by medical inspection in public schools and special studies in psychological clinics like the Witmer Clinic of the University of Pennsylvania.

6. Progress in tracing the intimate connection of mental defect with dependency, inefficiency and many social and economic maladjustments through the fruitful labors of numerous social workers.

7. Progress toward more effective supervision of immigration by trained psychiatrists in the United States Marine Hospital

service for the purpose of detecting mental abnormalities and excluding the insane, feeble-minded, epileptic and others likely to become public charges because of mental defect or insanity.

There is satisfaction that so much interest and activity in so many fields of inquiry have developed with little concert of effort, in response to obvious need, as seen by philanthropic persons with insight into the significance of fundamental causes. Sharp emphasis is thus laid upon the primary necessity of investigation as the preventive of hasty conclusions and guide to wise and effective action.

The great obstacle has been the scarcity of investigators of sufficient scientific instinct, capacity and training and the meagerness of facilities and funds for continuous and comprehensive research. The problems presented for solution are no less complex and exacting, no less needful of scientific scrutiny and accuracy of method, no less dependent upon many workers in many locations with encouragement of complete equipment and satisfying reward, no less hopeful of benefits to human welfare and amelioration of suffering, than the problems that have already been solved in other departments of medicine and science, where wonderful accomplishment gives promise of even greater attainment in the future.

The public should be aroused to consciousness of its obligation and self-interest in promoting adequacy in this direction. Every institution should do its part. The appeal to private benevolence is peculiarly strong. It may sustain the pioneer in the discovery of new principles and the test of new methods which, after demonstration of their truth and utility, will be accepted by the public and established in common practice.

There is great need of a brain institute for research into the nature, causes, results, prevention and treatment of mental disease and mental defect, established on a foundation as broad and comprehensive as the Rockefeller Institute for medical research in general.

Every large community needs its psychiatric hospital, free to the poor, the center of investigation and diagnosis of mental

abnormalities for the guidance of educators, juvenile courts and charitable agencies; for short intensive treatment of acute mental conditions on the plane of the hospital for physical diseases; in affiliation with the medical school and university for clinical teaching of psychiatry and abnormal psychology that the future family physician may be trained to foresee, prevent and afford early treatment of incipient mental disease; with its out-patient department to facilitate home treatment through a social service arm reaching out into the community to promote mental hygiene and supervise the after-care of the mentally affected who have been restored by treatment in institutions.

Public agencies usually lag far behind even in meeting the obvious requirements of demonstrated necessities. Private benevolence may lead the advance in investigation, in prevention, in curative methods which will ameliorate conditions and eventually reduce the public burden.

DISCUSSION

Dr. Francis X. Dercum: I do not think that this very interesting and timely paper of Dr. Copp should be allowed to pass without a word of comment. We all know how important the practical questions which confront us are in the field of insanity. We know the lamentable fact of the increase in the number that constantly demand our care in the hospitals, and also that they are greatly in excess of the facilities presented by the institutions now existing.

The method of dealing with the insane is a problem of great practical importance, and in this connection Dr. Copp has spoken of the psychiatric clinic and of the psychiatric hospital. There are many cases of recent or acute mental disturbance in which cure, a high degree of improvement, or long lucid intervals extending over many years, can be brought about and in which the patient can again be made an efficient or an approximately efficient member of the community. There are other patients, of course, who become impaired early and permanently; these require intelligent custodial care, a care so far as it can be made of retraining and of making the patients, as far as possible, contented and useful in institutional life.

After all, however, the victory in the therapeutics of insanity is to be gained by an increased knowledge of its pathology, notably of the role of internal secretions and of the autonomic and sympathetic nervous systems. A still greater victory is that of the future, for it lies in the prevention of insanity, and it consists in the diffusion through the community of the knowledge of the necessity for simple physiological and moral living. We find a great many cases of insanity with a history of syphilitic disease in the ancestry. Wassermann reactions for instance are found in almost one-third of the cases of dementia præcox. Alcoholism in the parents is another fruitful source of degeneration in the offspring. All causes which damage the germ plasm of the parent, of whatever nature, impair the structure and metabolism of the descendants. How great a role syphilis and alcohol play need hardly be pointed out.

We cannot, however, wait for the future but must deal with the problems which actually confront us at the present moment and how much can be accomplished with the insane now on our hands, Dr. Copp has many times demonstrated.

Dr. George E. Price: It is a curious thing that the branch of medicine which has to do with that most vital thing, reason, has been the slowest in scientific development. It is not so much more than one hundred years since the first attempt at rational care of the insane was inaugurated. This was introduced in France by Pinel, in England by Tuke, and in our country by Rush. Then came slowly the advance in the clinical knowledge of psychiatry, next research in the pathological field, and now it seems to me that Dr. Copp, in his excellent paper, has struck the keynote when he speaks of the movement toward prevention through education of the public. Certainly this can be brought about only as he said by, first, careful investigation. There is a great class of people whom Dr. Copp mentioned but did not emphasize, those who are apparently normal, yet who suffer because of an unstable nervous system due to the poor heredity which they have had. All men and women are not born free and equal in that respect. Many people because of this are forced to lead a more or less miserable existence. Inherent weakness handicaps them seriously in life's struggle. These are the cases referred to by Dr. Copp and Dr. Dercum who can be helped most by a proper understanding of the way in which they should live. Certainly Dr. Copp's paper is interesting and most timely.

Dr. Copp (closing): It seems to me that emphasis ought to be laid upon the necessity that a mental patient should receive the same study as any other case of disease. As an illustration of this, a school girl recently

came to notice who had developed some mental trouble, said to be dementia præcox. The medical inspector of the school regarded an underlying mental defect which existed as the essential condition, and advised commitment to one of the schools for the feeble-minded. When the case came before the Court the magistrate wanted further information. This was a very wise request which is not always made. The patient was, thereafter, received at the Pennsylvania Hospital and a thorough examination made, such as would be made in a general hospital. A positive Wassermann and spinal fluid were discovered. The investigation showed that we had a very interesting case of juvenile paresis. Further back in the history of the case it was found that the father died of general paresis in a hospital for the insane. The mother and five brothers and sisters, who submitted to the Wassermann test, were found to be syphilitic. The difference between the hospital attitude toward mental disease and the simple care attitude is this: In the latter, effort stops with care of the father; in the former, effort is made to prevent infection or bring the others under treatment.

There is a great deal to be done for mental patients. They do get well; not, perhaps, as large a percentage as we would wish, but during the last year 55 per cent. of a large admission rate at the Pennsylvania Hospital went home recovered or capable of living under the ordinary conditions of home life.

Regarding the chronic patient, it is not only not humane, but not economical to neglect him. Much benefit follows proper treatment and study. There is possible partial regeneration of nerve centers and development of dormant energies which can be diverted into useful channels, thus reducing the cost of care of the patient, and contributing materially to his support. The matter is not one about which we should be indifferent, but rather one which we should attack.

A BABYLONIAN PHYSICIAN OF 2400 B.C.[1]

By MORRIS JASTROW, Jr., Ph.D., LL.D.

The Louvre Museum possesses in its Babylonian collection a seal cylinder[2] showing, as part of the design, what I take to be medical instruments—apparently two knives and a scalpel—and in addition two jars, resting on high stands, which may be intended for ointment pots. The inscription accompanying the design reveals the name of the owner of the seal, who calls himself Ur-Lugal-edinna, and adds as his profession *a-zu*, *i. e.*, "the physician." The general form of the characters of the inscription indicates that this physician flourished in the old Babylonian period, but his exact date could not be determined from this indication. Quite recently, however, in an important volume of Cuneiform Texts, published by Professor A. T. Clay, of Yale University, and containing some fifty texts, there is found a votive inscription addressed to the Babylonian goddess Bau,[3] which reads in translation as follows:

> "To Bau, his lady, for the life of Ur-Ningirsu, the *patesi*[4] of Shirpurla, Ur-Lugal-edinna, the physician, and for his own life has presented."

There can be no doubt as to the identity of the one who dedicates this inscription with the physician named on the seal. Not only are the names written alike, but in both cases the profession is added. The characters of the votive inscription are old Baby-

[1] Presented by Dr. Keen at the meeting of April 5, 1916.

[2] See De Sarzec, Dècouvertes en Chaldèe, Pl. 30, bis 16*a* and 16*b*, and pp. 301–303, and Ward, Seal Cylinders of Western Asia, p. 255, where a full description of the cylinder will be found.

[3] Yale Oriental Series, Babylonian Texts, vol. i, No. 8.

[4] Title of the rulers of Shirpurla.

lonian, precisely as upon the seal, and to clinch the matter, Shirpurla, the city and district over which Ur-Ningirsu ruled, is the ancient name of the mound Tello in the course of the excavation of which by the French Assyriologist, the late Ernest de Sarzec, our seal cylinder was found. The votive inscription therefore confirms that Ur-Lugal-edinna was a physician who practised in the ancient city of Shirpurla in southern Babylonia, the center of one of the important States or petty kingdoms into which in the earliest period the Euphrates Valley was divided. Moreover, by the mention of Ur-Ningirsu as the ruler (or *patesi*), for whose life the physician dedicates an inscription (no doubt

accompanied by an offering to the goddess Bau), and who is well known to us from inscriptions that have likewise been found at Shirpurla, the date of Ur-Lugal-edinna can be fixed at about 2400 B. C.[1] Ur-Lugal-edinna, thus fixed as a contemporary of Ur-Ningirsu, turns out to be the oldest Babylonian physician known to us by name; and we may furthermore conclude that inasmuch as he dedicates an inscription to the ruler, he must have held an official position, no doubt that of court physician. I had hoped to be able to add that Ur-Lugal-edinna was also the oldest physician

[1] Thureau-Dangin, Sumerisch-Akkadische Koenigsinschriften, pp. 146–148, and King, History of Sumer and Akkad, p. 274, *seq.*

known to us by name in the world. Egypt, however, is ahead of Babylonia in this respect, for we find a certain Imhotep mentioned in Egyptian monuments as court physician of King Zoser, who belongs to the early portion of the Third Dynasty of Egypt, and which, according to the chronology at present accepted by Egyptologists, would place Imotep at about 3000 B.C.[1] I might add, in explanation of the name of the Babylonian physician, that Lugal-edinna is the name or title of a deity, and that the element Ur means "man" or "worshipper."

[1] See for a notice of this physician who was so venerated that he was finally worshipped as a god of medicine, identified by the Greeks with their own Asklepios, Breasted, History of Egypt, p. 112, *seq.*

DERMATITIS CAUSED BY COSMETICS AND WEARING APPAREL, PARTICULARLY THOSE CONTAINING PARA-PHENYLENE DIAMIN[1]

By FRANK CROZER KNOWLES, M.D.

The cause of disease particularly from the dermatological point of view is frequently an unfathomable problem notwithstanding the numerous valuable investigations which have been instituted. It is surprising how frequently we are close to the solution of the problem and an illusive something prevents the elucidation. Care in research, however, each year gives us more etiological data. This is truly exemplified in the realization of the resistance, really lack of resistance of the skin, to certain irritants, particularly to those at present under discussion. It has been of only recent years that dermatitis of the scalp, the face, the neck, and the wrist could be positively ascribed to irritants the exact formulæ of which is known. Women are particularly prone to these forms of outbreak because they depend more generally upon cosmetics and personal adornment.

Mewborn, in 1901, reported a case exhibiting a severe dermatitis of the face, in which the forehead was swollen, red and shiny, with a few small vesicles near the margin of the hair. The eyelids, particularly the upper, were puffy, the conjunctivæ slightly congested, the ears red and swollen, and numerous small vesicles, many of which had broken with the formation of crusts, were observed along the margin of the affected areas. The nose and

[1] Read May 3, 1916.

the cheeks were slightly swollen. The majority of the outbreak was noticed along the hair margin and on the upper part of the ears. The itching was intense. The face felt tense, uncomfortable, with some prickling sensation, especially marked in the eyes.

Mewborn discovered that she had been using a French hair-dye, which had given rise to the eruption a few days after starting its use. The hair-dye was found to depend for its action upon paraphenylene diamin, a substance which when oxidized by means of a solution of hydrogen dioxid is converted into quinone, and with which tints varying from auburn to jet black may be produced.

An aqueous or alcoholic solution of the diamin is first brushed or sponged on, and a few seconds later oxygenated water is similarly applied, giving rise to an immediate dying. Unfortunately quinone sublimes at comparatively low temperatures and gives off most irritating vapors, which are apt to excite a dermatitis of an erythematous character, with swelling, papular and vesicular lesions.

Apparently the major portion of hair-dyes, although marketed under various titles, depend for their action upon paraphenylene diamin. The French observers have until recently reported the greatest number of cases of dermatitis from this substance; Cathelineau alone recording eighteen examples. The frequency of occurrence of the eruption from this chemical can be accurately judged when eight instances of the affection were reported in the *Journal of the American Medical Association* during the first six months of 1909. Scores of these cases have been seen, many not having been officially recorded. Heimann has reported within the last month six instances.

The distribution of a dermatitis in the upper third of the face, the swollen eyelids, the vesiculation of the rims of the ears are suggestive of the cause. The reaction frequently occurs several days or even weeks after the last application of the dye.

According to Chipman the eruption begins as an erythema varying doubtless with the resistance of the skin as well as the strength and frequency of the application. He states that following the

erythema, an edema of the skin is observed, and later a desquamation which is proportionate to the intensity of the original erythema. Zeisler has seen quite a number of instances of dermatitis during the last few years due to hair-dye most of which cases were observed in women above middle life, one only occurring in a man aged sixty years. He mentions that a close observer will quickly notice the unnatural dead color of the hair, which contrasts markedly with the grayness of the hair close to the scalp. The hairs for a fraction of an inch at the proximal ends, close to the scalp, are of a different tint from the remaining portions. Morrow records the fact that the eruption produced by this chemical sometimes persists for six to eight weeks after the last application and he knows no other agent which when applied to the skin produces such a persistent eruption.

Chipman believes that the irritating substance is spread mechanically by the common practice of women combing their hair forward and downward over the face. Foerster states that in his experience, within a few hours after the application of the hair-dye the individual notices the sensation of heat and burning confined to the scalp. After some hours, frequently during the night after the hair has been for some time pressed down closely to the head by the pillow, the eruption makes its appearance at the hair margin and spreads to the contiguous areas. It is thought that in the cases exhibiting fairly generalized dermatitis the irritating vapor is carried to the affected areas by the hands.

A. Gautier has written an exhaustive work on the chemical constituents of hair-dye, and he points out that in certain instances a single application of paraphenylene diamin has sufficed to induce a toxic action. The symptoms of intoxication from this and other poisonous anilin dyes may be divided into three groups: (1) Toxic skin eruptions, dermatitis and urticaria, with great burning and itching; (2) gastro-intestinal symptoms, such as nausea; (3) nervous symptoms, sleeplessness, dizziness, weakness of the legs, epileptiform attacks and syncope. In several instances death has resulted. Cases have been reported in which retrobulbar neuritis, with impairment of central vision, and a central

scotom for red and green were observed. Damianos has recorded an instance of chronic poisoning from a hair-dye containing this substance which was employed over a considerable period. The patient exhibited chronic nervous exhaustion, dread, weakness, vertigo, photophobia and recurrent clonic spasms which finally became so severe that the woman was confined to bed in a dark room.

Paraphenylene diamin, however, is not the only chemical which is responsible for a dermatitis of the scalp and the contiguous parts. Scharff has recently reported an eruption caused by a Javel hair lotion. Resorcin in strong solution, particularly in the susceptible individual and when thoroughly rubbed in, not only may produce a mahogany color change, mostly in the blond and white-haired individual, chiefly when the head is exposed to the summer sun, but may also produce a marked dermatitis, with numerous miliary vesicles. The scalp is intensely red and there is oozing, edema, and frequently a like condition of the upper portion of the face. The eyelids may be markedly swollen and the patient has just as marked an outbreak of the face as is seen in exaggerated cases of ivy or oak poisoning.

Along somewhat analogous lines, Lezinsky has recorded an instance of a dermatitis of the face produced by the use of a "triple extract of heliotrope" employed as a toilet article. Foerster reported a puzzling series of cases, in which there was moderate swelling and stiffness of the lips, particularly the upper, confined to the mucocutaneous surface, with persistent exfoliation in small flakes, and slight fissuring accompanied by marked tingling and a burning sensation most marked in the morning. The outbreak was caused by a proprietary mouth wash which contained formalin. Heimann recorded an interesting case in which a dermatitis was caused by a bust developer, the outbreak extending to the extremities, neck and the face.

Dermatitis due to wearing apparel is of frequent occurrence and therefore an etiological factor should always be sought. Stockings and undergarments dyed with picric acid, chromium or arsenic are often causal of an eruption. The leather linings of

hats or caps are prolific sources of an outbreak. Duhring reported cases in which the dyestuffs in the lining of shoes penetrated the material in the stockings of women and produced dermatitis of the feet and legs. Aurantia or hexanitrophenylamin which is used for the staining of cheap yellow leather shoes may in a like manner cause an outbreak.

Anilin dyes, particularly the red ones, and J. C. White states the black ones also, are often causal of an eruption, chiefly through clothing such as gloves, socks, flannel shirts, drawers, etc., dyed with these substances. They are apt to excite an itching, papular eruption, which in extreme cases become of a vesicular or pustular type. Though, at first limited to the parts in contact with the dye, the eruption often extends to a considerable distance beyond the part first affected. Accidental contamination of the dye with arsenic is supposed to be the real cause of the outbreak, but some ascribe it to the anilin itself.

Comparatively recently articles have been appearing upon the dangers of using dyed fur because of the tendency toward a dermatitis. The present extensive use of dyed fur, especially for collars has caused many cases of painful dermatitis. Although arsenic used in preparing fur may occasionally cause a dermatitis, practically all of the cases are produced by the wearing of brown or black fur dyed with paraphenylene diamin. This substance is an allied and homologous compound of anilin black, and is oxidized by hydrogen peroxid and other chemical substances to form Bandrowski's base. The intermediate product of oxidation, according to Olson, is chinondichloridiimin, which is very irritating and is the cause of dermatitis from dyed fur. Where oxidation is complete, paraphenylene diamin is not irritating, and this probably explains why the brown fur is more irritating than the black.

Olson states that the brown fur collars worn by men and the black pony worn by women are particularly to blame for the outbreak. He also mentions that the deeper part of the hairs in the fur show a very characteristic, brick-red color, even though the fur may superficially appear to have a natural brown color. In his 5 cases he found that the "collar area" was the part of

predilection for the outbreak, next the hands, and in certain instances the dermatitis may extend generally. Some persons are apparently not susceptible to this dye and develop no symptoms, even though the chemical in the fur is rubbed off and darkens the skin to a noticeable degree. Some individuals are not ordinarily affected by wearing these dyed furs, but an acute outbreak may be excited, provided the skin happens to be especially sensitive, as after a close shave, or if moisture comes in contact with the fur, as from melted snow, or in extremely cold weather from precipitation of the moisture in the breath. In other words, any factor causing the fur to become damp increases materially its toxic properties. Hartzell has mentioned cases of dermatitis from the wearing of fur boas. Foerster has recorded an instance of the outbreak of an eruption on the wrists and the neck from the fur of an overcoat coming in contact with these areas. Jas. C. White, thirteen years ago, reported instances of dermatitis of the face and the neck from wearing fur tippets.

The writer has seen 3 cases of a characteristic dermatitis caused by hair-dye, all in women past the age of forty years, and the same number of instances, also in women, of an outbreak from the wearing of brown or black fur around the neck. Several patients have exhibited a resorcin dermatitis extending downward from the scalp. Examples of outbreaks on the face have also been treated, caused by various cosmetic skin foods or face creams. In some of the cases of resorcin dermatitis, the question naturally arises as to the amount of influence friction used in applying the preparation might have had upon the induction of the outbreak, in others, however, the lotion has been applied with a dropper, and massage was not employed.

The query would be thought of as to why so many individuals are apparently immune to an outbreak from paraphenylene diamin. This can only be explained by a certain resistance on the part of an individual or else that the dying process has been more carefully carried out and some of the irritating factors removed. The fact that certain persons are not affected for some time, even months, after constant exposure to this dye has been explained

on the assumption of an anaphylaxis, or of an increasing susceptibility, or because of the accumulative effect of the toxic properties of the dye.

This hair-dye is prohibited by law from being sold in France, Austria and Germany, and therefore the toxic properties of the chemical can be realized. Olson makes the reasonable suggestion that if the selling of fur dyed with paraphenylene diamin is not prohibited by law, there should at least be a label attached stating that this dye has been employed.

The custom of wearing fur is so universally decreed by fashion that the laiety in general should be cognizant of the dangers of an outbreak. Physicians should be constantly on the *qui vive* for this cause of dermatitis. It should be recognized as a prophylactic measure that dyed fur should not touch the skin. Discarding of the causative factor should be insisted upon in the event of dermatitis.

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THE VALUE OF SPLENECTOMY IN THE PRIMARY ANEMIAS*

By EDWARD B. KRUMBHAAR, Ph.D., M.D.

Removal of the spleen for therapeutic purposes is one of the oldest abdominal operations about which we have definite knowledge.[1] Undoubtedly practised by the Greeks and Romans, it was continued at rare intervals through the Middle Ages, as is shown, for example, by the much-quoted operation of Zaccarelli, performed in 1549. Although it was early known that the spleen was not necessary for life, splenectomy was at first done only in the case of rupture or severe injury of the organ. Eventually, however, with the advent of anesthesia and the greater surgical skill of the nineteenth century, it was found practicable to remove the chronically diseased organ, and thus widen the field of applicability of the operation beyond that of surgical emergencies. Unfortunately, among the chronic diseases of the organ first attacked were the enlargements incident to cirrhosis of the liver and leukemia. The unfavorable results in these two diseases cast discredit upon the operation; but, nevertheless, in 1900 Bessel-Hagen[2] was able to collect 216 cases of total extirpation of the spleen with 72 deaths. If the cases of leukemia are subtracted, the deaths in the list are reduced to 34, or a mortality of 15.5 per cent. Since 1900 our greater knowledge of the physiology and pathology of the spleen has resulted in a better selection of cases, so that now the total mortality has been still further reduced (Laspeyres[3] and Michelson[4]).

* Read May 3, 1916.

In the past three years a more active study of the surgical treatment of certain so-called primary anemias has led to the much wider application of splenectomy, and it is this application of splenectomy that attracts most attention at present.

Contra-indications. It is most important to know when splenectomy should not be done. We now know that in certain diseases, removal of the enlarged spleen is definitely contraindicated. These include the various forms of leukemia, polycythemia, malaria, atrophic cirrhosis of the liver, and most cases of tuberculosis and syphilis. In the hypertrophic form of cirrhosis Eppinger has recently advocated splenectomy on account of the evidences of increased blood destruction in this disease, but in the ordinary atrophic forms it certainly should not be considered. Too much emphasis cannot be laid on the necessity of ruling out atypical forms of leukemia before the splenectomy is undertaken, but on account of the great variety of aleukemic conditions this is often an extremely difficult task. In no case, however, should splenectomy be advised until the blood picture has been carefully studied over an extended period of time, and the presence of leukemia excluded as far as may be possible.

Anything pointing toward a hemorrhagic diathesis should also be given careful consideration. Its presence is, as a rule, sufficient to contraindicate operation, although the repeated hemorrhages from varices due to other mechanical causes, as in Banti's disease, are more indications for operation than otherwise.

In the severer anemias definite signs of bone-marrow activity should also be forthcoming (nucleated or reticulated cells, Jolly bodies, etc.). If they cannot be provoked by appropriate drugs or by transfusion it is probable that the marrow is aplastic and splenectomy should not be attempted.

That the removal of the normal spleen is followed by a temporary anemia has been proved both by clinical observation and animal experimentation, but this should not be considered a contra-indication to operation. We have the apparent paradox that while removal of the normal spleen causes a temporary anemia, removal of the spleen in certain blood diseases relieves the existing anemia.

Diseases in which Splenectomy is Indicated. In certain groups of disease, splenectomy has met with considerable success: as in Banti's disease, Gaucher's disease, the congenital and acquired forms of hemolytic jaundice, and, to a lesser extent, pernicious anemia. The cause of the improvement or cure that follows splenectomy in these conditions is but poorly understood. It is probably different in the various diseases mentioned, but a discussion of this problem cannot be undertaken here.

Table I.—Collected Reports of Results of Splenectomy in Blood Diseases

Disease.	Author.	Cases.	Recovered.	Died.	Mortality, per cent.
Gaucher's . . .	Erdmann and Moorhead[5]	10	8	2	20.0
Banti's . . .	Isaac[6]	49	41	8	16.3
Hemolytic icterus:					
acquired . .	Elliott and Kanavel[7]	16	15	1	6.2
congenital and familial . .	Elliott and Kanavel[7]	23	22	1	4.3
unclassified .	Elliott and Kanavel[7]	9	9	0	0
v. Jaksch's . .	Graff[8]	2	2	0	0
Pernicious anemia	Krumbhaar[13]	153	123	30	19.6

Banti's Disease. In Banti's disease it is important that the operation should be undertaken before the disease has progressed beyond the first stage of the disease. Splenectomy in the first stage is not only accompanied by a low mortality, but in the great majority of cases has caused great and lasting improvement of symptoms, often amounting to a complete cure. When the third stage is reached, with permanent changes in the liver and circulatory system, not only is the operation more dangerous, but the chances of improvement are greatly lessened. As a result of the disease process, the spleen has by this time become largely fibrotic, and its removal could hardly be expected to be attended with marked beneficial effect. Cases have been reported, however, where improvement followed splenectomy even at this late stage.

In 1912 Isaac[6] collected 49 cases with a mortality of 16.3 per cent. With the better understanding of the disease and its treatment that has come about since that time it is safe to say that

the mortality percentage for Banti's disease is now considerably lower. A patient, then, with the symptoms of Banti's disease, particularly if in the early stages of the disease, should be considered a proper candidate for splenectomy, but the most favorable time for operation should be selected. Before undertaking the operation, all other possible causes for such a syndrome (*e. g.*, aleukemia, leukemia, tuberculosis, malaria, syphilis, etc.) should be ruled out as far as possible by a complete, but not unnecessarily prolonged, investigation as to the cause of the disease, with frequently repeated blood examinations.

Gaucher's Disease. On account of the rarity of this condition and the difficulty of diagnosis without the aid of histological examination, not many cases are available for study. In 1914 Erdmann and Moorhead[5] collected 10 cases of large-celled splenomegaly (Gaucher's disease) in which the spleen had been removed, and of these 2 died, both within twenty-four hours of operation. While this probably represents too high a mortality, the improvement which followed in the other 8 cases cannot always be taken as indicative of eventual cure, for the disease is known to exist independently in the bone-marrow and lymph nodes. It would therefore seem wiser to restrict splenectomy in this disease to those cases that are unusually handicapped by the results of the disease but are still good surgical risks, and in such cases to limit the prognosis to improvement but not complete cure.

Hemolytic Jaundice. The field in which splenectomy has been practised with the greatest success is undoubtedly that of hemolytic jaundice. Both in the acquired form (Hayem-Widal) and the congenital or familial type (Chauffard-Minkowski) marked improvement and frequently complete cure has resulted from removal of the spleen. In fact, the success obtained in this type of case, in which the chief vitium is that of increased blood destruction, has been a powerful incentive toward extending the operation of splenectomy to the wider range of allied diseases discussed in this chapter. Splenectomy was first recommended in this group by Banti for a condition to which he had given the name "hemolytic splenomegaly." Success was obtained in all the

earlier operations (Banti, Micheli, Kahn, Roth), and the procedure would undoubtedly have been in more frequent and intelligent use, in this country as well as Europe, if the several clinical entities unfortunately grouped together under the cloak of "splenic anemia" had not clouded the worth of the procedure. In 1915 Elliott and Kanavel[7] were able to collect 48 cases of hemolytic jaundice (16 acquired, 23 familial, and 9 unclassified) that had been treated in this way. Of the 48 cases, only 2 died, 1 shortly after operation, the other from sepsis, six weeks after operation. The other 46 are reported as "cured," this result being based upon the disappearance of jaundice and exacerbations, and decrease of the anemia and the urobilin excretion. The effect on the resistance of the red cells was not constant; in some instances the resistance returned almost to normal, but in most cases the red cells remained almost as fragile as before operation.

Pernicious Anemia. The most important disease to which splenectomy has been applied, from the point of view of its greater frequency and greater severity, is pernicious anemia. The first attempts were made independently by Eppinger and von Decastello in Vienna in 1913 and were shortly followed by Klemperer in Berlin in the same year, and soon after, on account of the initial improvement observed, by a large number of surgeons all over the world. Although it is still too early to proclaim any definite decision as to the value of splenectomy in pernicious anemia, it has already become possible to base opinions on substantial evidence. I have recently collected the results in 153 cases treated in this manner. In thirty instances death occurred either immediately or within six weeks of the operation. Of those surviving the operation, nearly all showed a marked improvement both clinically and in the blood picture. The so-called "blood crisis" (appearance of numerous nucleated red cells and Jolly bodies), indicating a more active bone-marrow, usually was observed within a day or two of the operation, and was followed for several weeks or months by a steady rise in hemoglobin and red blood cell count. In a few cases this increase, coincident with gain in weight and improvement in the patient's strength and general condition,

has persisted during the period of observation—in 6 cases over a period of two years. In the majority, however, the blood picture of pernicious anemia remained, and after several months of improvement the patient returned to the same condition as previous to operation. Of 27 individuals that were living at the end of the first year after operation, 9 died later, 11 were still improved and 7 have relapsed to the preoperative condition, while 6 showed continued improvement at the end of two years. It is noteworthy, though not surprising, that those cases presenting spinal-cord changes show no improvement in nervous symptoms, and are otherwise less improved by the operation than are those cases in which spinal-cord changes do not exist.

Although great improvement, therefore, may be said to persist in a certain number of cases, in the majority the effect of the operation is to produce only a remission, such as is characteristic of the natural course of the disease. When, then, should the operation be performed? Should it be withheld, as a trump card, to stave off death for several months when all other measures have failed; or bearing in mind the minority cases that acquire more lasting improvement, should it be done as soon as a sure diagnosis is made, before the disease has progressed too far or spinal-cord changes set in? Also, should every case of pernicious anemia have the spleen removed? Certainly, in such cases as those reported by Antonelli[9] and Mosse,[10] which might easily be considered cases of hemolytic jaundice, with splenomegaly, increased fragility and urobilin output, better results should be expected than in cases at the other extreme of the group, with no demonstrable signs of increased hemolysis, and perhaps an aplastic bone-marrow. Statistics also show that better results are obtained if the operation is preceded by transfusions, if the patient is under fifty years of age, and has not had the disease for many months. The presence or absence of spinal-cord changes may also prove to be an important factor. Such questions, however, must remain unanswered until the lapse of time and acquisition of more evidence will allow us to decide conclusively. The operation has already gained sufficient vogue to allow one to

predict that such an accumulation of evidence will soon be forthcoming.

Differential Diagnosis. The differential diagnosis of chronic splenic disease is not only important in the selection of proper cases for splenectomy, but also in order that a more accurate prognosis may be given. For the details of the differential diagnosis the reader is referred to other text-books and medical articles,[11] but the following points may be alluded to:

The characteristic picture of Banti's disease is that of a disease running a chronic course, usually occurring in otherwise healthy young adults, and divided into three periods. In the first, or pre-ascitic period, usually lasting several years, a gradually increasing weakness and pallor is noticed with digestive disturbances and abdominal pain, which may first call attention to the enlarged hard spleen. A tendency to hemorrhages with a moderate anemia of the chlorotic type is usually present. There is nothing characteristic of this anemia, an increased urobilin excretion being the only sign of increased blood destruction. The resistance of the red cells is unchanged, and signs of a regenerating bone-marrow (nucleated and reticulated red cells) are slight or absent. Leukopenia is usually present. The second or intermediate stage, characterized by scanty urine, attacks of diarrhea and dyspepsia, and increase in size of liver, lasts but a few months, and is usually merged in the general picture. The features of the third stage are cirrhosis, recurrent ascites and jaundice, and increasing emaciation and anemia.

In Gaucher's disease the onset is more insidious, the symptoms appear in infancy or childhood, and the disease runs a more chronic course. A history of similar trouble in the family is frequently elicited. The huge size of the liver with attending abdominal discomfort is the most prominent symptom, and histological examination either by splenic puncture or after splenectomy reveals the large vesiculated cells with small eccentric nuclei characteristic of this disease. A brownish discoloration of the skin, with "peculiar yellowish wedge-shaped thickening of the conjunctivæ" on both sides of the corneæ, has been noted. The

TABLE II.—DIFFERENTIAL DIAGNOSIS

	Etiology.	Family history.	Time of onset.	Duration.	Icterus.	Splenomegaly.	Hemorrhages.	Anemia.	Resistance of red blood cells.	Reticulated red blood cells.	Urobilinuria.	Leukocytes in blood.	Liver.	Treatment.
Gaucher's disease	Toxin? Infection? Tumor?	Occasionally positive	Childhood	Many years	Rare	+++	Occasionally	Slight	?	?	?	−	+	Splenectomy?
Banti's disease	Toxin? Infection?	Negative	Adult life	Few years	Rare	+	Occasionally	Slight to severe	Normal Normal	Normal Normal	+ +	− −	First + Later −	Splenectomy.
v. Jaksch's disease	Rachitis?	Negative	Infancy	Several months	Rare	++	?	Slight to severe	?	?	?	+	+	Splenectomy? Iron, arsenic, etc.
Hayem-Widal	1. Primary? 2. Secondary to infection	Negative	Any age	Many years	Present	++	Rare	Severe	Diminished	Increased	+	−	+ or normal	Splenectomy.
Chauffard-Minkowski	Heredity	Positive	Congenital or childhood	Many years	Present	++	0	Slight	Diminished	Increased	+	−	+	Splenectomy.
Pernicious anemia	Enterogenous toxin?	Negative	Adult life	Few yrs. with remissions	Very rare	Slight or diminished	Rare	Severe	Increased	Increased	+	− or +	Normal	Iron, arsenic, etc. (splenectomy questionable).

changes in the blood are similar to, but less marked than, those in Banti's disease.

In hemolytic jaundice the characteristic features are the persistent acholuric jaundice, the enlarged spleen and the decreased resistance of the red blood cells to hypotonic salt solution (normally hemolysis begins at 0.45 per cent. NaCl and is complete at 0.35 per cent.). This is usually accompanied by enormous increase in urobilin output and in the percentage of the reticulated red cells. The number of nucleated cells depends on the degree of anemia, which tends to be severe in the acquired form and mild in the congenital or familial type. Crises characterized by increased severity of symptoms and anemia are common to both types.

As von Jaksch's pseudoleukemia is probably not a clinical entity and pernicious anemia is such a well-known condition, they need not be dealt with at this time.

Choice of Time for Operation. On general principles it may be said that splenectomy should be undertaken as soon as the diagnosis is definitely settled. In certain instances, however, exceptions must be made. A "crisis of deglobulization" in hemolytic jaundice or a severe hemorrhage from a mucous membrane in Banti's disease would indicate postponement of the operation until the usual condition (for the disease) of health is regained. The existence of a possible contributing factor, as indicated, for example, by the finding of a positive Wassermann reaction or of malarial organisms, would naturally postpone operation until the influence of such factor had been, as far as possible, eliminated. When the anemia is severe, and this applies particularly to pernicious anemia, a series of blood transfusions (often four or more) should precede operation. If the patient's blood in this way is temporarily enriched, not only is the operative risk lessened but more lasting benefit ensues. Good results have been obtained in pernicious anemia by the subcutaneous or intraperitoneal injection of splenic extract,[12] and in cases in which the so-called "blood crisis" fails to materialize after splenectomy, indicating a failure of the bone-marrow to respond, this procedure might be tried.

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DISCUSSION

Dr. Edwin E. Graham: In a consideration of the prognosis of Banti's disease, Dr. Krumbhaar has divided the cases into three series, first, second, and third, according to the severity of the case.

It would, I am sure, be of interest if he could give us some information as to the difference in the prognosis in the three grades of this disease, as he has distinguished them.

The question of the advisability of splenectomy depends largely upon the stage of splenic anemia in which the operation is performed; if done early, the prognosis is favorable; if performed late, when cirrhotic liver, ascites, and jaundice have developed, the prognosis is much less favorable.

Dr. Krumbhaar (closing): I am afraid I cannot answer Dr. Graham's question much more definitely than I have stated in my paper. Isaac's figures of 1912 are already a little antiquated, and although they are the latest available, undoubtedly many more cases of Banti's disease have by this time had their spleens removed. I recall one individual's record in which very good results were found after splenectomy was done late in the third stage, when the patient was very anemic, and with an advanced degree of cirrhosis and ascites. Further than that I could not say.

(N. B.—Later investigations have brought out 20 reported cases of Banti's diseases that had had their spleens removed in the third or ascitic stage. Of these 4 died soon after operation, but most of the others were reported as improved. Of 70 odd cases operated upon since 1910 the mortality was just over 10 per cent.)

GLUCOSE FORMATION FROM PROTEIN IN DIABETES*†

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Introduction. The origin of carbohydrates from protein in the animal body has become a very firmly established fact in spite of the animated controversies of the past. Proteins have been demonstrated in numerous researches to yield carbohydrates in metabolism. The same holds true for many of the amino-acids, which enter into the constitution of proteins.[1] Indeed, there is good existing evidence showing that this is to be regarded as a normal metabolic process.[2]

It is, however, in the study of diabetes that a knowledge of sugar formation from protein becomes very essential, for not only is protein food ingested by diabetics converted into glucose, but extensive formation of this monosaccharid is known to take place from the diabetic's own tissue proteins. The importance of these considerations has stimulated numerous experimental investigations. Although much new data have been obtained as a result, considerable obscurity still remains. This applies especially to the question of the exact extent of the conversion of protein into glucose, a weighty subject for a clear conception, both of the diabetic process and of diabetic dietetics. In this laboratory

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† Preliminary reports of the experiments detailed in this section were presented in papers read before the Society for Experimental Biology and Medicine, January 19, 1916, and the College of Physicians, Philadelphia, June 7, 1916.

during recent years considerable time has therefore been devoted to its study. It is the purpose of the present communication to call attention to the results of medical importance obtained in this work as well as to report further experimental data bearing on the subject of the dietary of diabetes.

Critique of Former Investigations. Previous efforts to ascertain the amount of glucose arising from the break-down of proteins in metabolism have met with so many experimental difficulties that in almost all cases the results obtained are open to serious criticism. It seems therefore expedient rather to emphasize the various sources of error in this work than to indulge in an elaborate critical review, which has, indeed, already been done by others[3] who also quote the literature extensively. The human diabetic has been in the past the subject most frequently selected for experiments of this nature.[4] Various proteins were fed to glycosuric patients and the influence on the sugar excretion observed. The results showed a rise in the glycosuria following the ingestion of protein, but very little certainty as to the extent of either absolute or relative sugar production from protein can be attached to this work, for reasons to be stated.

In order to ascertain, by means of feeding experiments, the extent of glucose formation from ingested proteins it is necessary to demonstrate that all the fed material is digested and absorbed and that all the glucose arising from this material, and no more, originates from this protein. These conditions can hardly be afforded by the human subject, as will now be brought out. In most cases of diabetes mellitus a certain amount of oxidation of glucose takes place. Moreover, the capacity of the human diabetic to utilize glucose frequently may undergo considerable daily variation, even when the diet remains the same. It is also possible that glucose originating from food protein may be in part synthetically used in the formation of various body substances or be deposited as glycogen. In regard to these last considerations there are very few definite data as yet on record for the human diabetic.

The reliability of such metabolic experiments is dependent on the complete assimilation of the protein fed. In the former

experiments referred to, this important matter has not been adequately controlled. The important physiological factors in this regard will be discussed later in this communication. It may suffice to mention here that the completeness and rapidity of absorption of protein is dependent not only on the amount and physical condition of such food, but also on the nature of other constituents of the diet. Experiments on human diabetic individuals, in which a given amount of protein is superimposed on other diet, are therefore rather uncertain.

It would thus seem advisable to use fasting diabetics for such experiments. The effect of starvation on the human subject is, however, as is so well demonstrated by Allen's researches, to immediately increase the ability of the organism to oxidize glucose. We cannot then expect glucose arising from ingested substances to be excreted quantitatively by otherwise fasting diabetics. This is undoubtedly a source of error in certain human experiments already reported, in which protein has been administered to patients after days of greatly restricted diet. Another and contrary effect of feeding quantities of sugar-forming proteins to diabetics is to lower the tolerance of the organism for glucose, with consequent uncertainty in the results. This is very evident in certain of Mohr's experiments. Still another disturbing factor in the use of the human diabetic is the fact that muscular exercise may decrease the glycosuria under some circumstances, but increase it under others.[5] But the great difficulty of preventing diabetics breaking diet is very probably the chief cause of error in such human experiments.

Through one or more of these sources of error the results of protein-feeding experiments and clinical observations are rendered dubious. Conclusions drawn from these results cannot therefore be regarded as established. Among such may be mentioned those of Lüthje, von Noorden, and Falta, who hold that relative differences in the influence of protein food on the glycosuria are observable in light or moderate cases of diabetes. Falta's observations that those protein foods which are more difficult of digestion more favorably influence the glycosuria also illustrates the diffi-

culty of such experimentation. If the alimentary factors had been carefully controlled in these experiments, it is probable that very different results would have been obtained.

In endeavoring to avoid these difficulties the depancreatinized diabetic dog also has been made use of by other workers. Unfortunately such animals are ill adapted to fine metabolic examinations of this type, for the reason that extirpation of the pancreas is followed by very severe effects, which are indeed particularly marked in case of the digestive system. It is surprising that no certain results have been obtained by this method of experimentation. There is also considerable evidence which cannot here be detailed, to the effect that a certain amount of glucose utilization does take place locally in depancreatinized dogs in spite of data to the contrary.

Glucose formation from ingested protein has also been studied experimentally in a few isolated instances with the aid of dogs made diabetic with the use of the glucosid phlorizin. The work of Halsey,[6] Rohmer[7] and Bendix[8] may be referred to. The technic employed is open to various criticisms, which preclude drawing definite conclusions from this work. Reilly, Nolan and Lusk,[9] however, in two experiments were able to determine the amount of glucose formed from gelatin in metabolism.

Development of Improved Experimental Methods. As both the human diabetic and the depancreatinized dog represent serious obstacles to the successful study of glucose formation from protein, recourse was made to phlorizin diabetes. Although many experimental difficulties were encountered here also, none proved insurmountable, and a technic could be developed by which the extent of protein conversion into glucose could be followed with considerable accuracy. The various details bearing on this point have been dealt with in a preceding series of articles,[10] so a repetition scarcely seems indicated. A few main points, however, may be alluded to, as the nature of phlorizin glycosuria is not so well known to many as are other forms of melituria. This kind of diabetes has been very carefully studied by Lusk and a number of other workers within recent years with the result that its metab-

olism is now fairly adequately understood. When phlorizin is administered to dogs in the proper manner, complete diabetes rapidly develops, the reserve of carbohydrates within the body is quickly exhausted and in the fasting state the glucose appearing in the urine bears a constant relation to the urinary nitrogen, this so-called glucose-nitrogen ratio averaging 3.4 to 1.[11] Glucose administered to such animals within certain limits is quantitatively excreted.[12] Glucose arising from non-toxic ingested substances fails to be stored up or elsewhere utilized, but appears in the urine also quantitatively. This subject has been discussed previously by the writer, who has detailed the reasons for accepting this view.[13] Recently experiments from this laboratory have been reported, which also make it very probable that all the glucose arising from protein fed to phlorizinized dogs is excreted in their urine.[14] This work demonstrated that the urinary glucose and nitrogen of fasting phlorizinized animals, which quantitatively excrete ingested sugar, bear the same relation to each other as the extra glucose arising from these animals' own proteins ingested by other phlorizinized dogs does to the nitrogen contained in these proteins. In other words, just as much glucose appears in the urine after ingestion of phlorizinized dogs' proteins as is yielded by the break-down of the fasting animals' living protoplasm. Such identical results would be impossible if all the glucose originating from the ingested proteins did not appear in the urine, for we have reason to believe that carbohydrates are not stored by fasting phlorizinized dogs (Lusk). Consequently the sugar excreted represents the maximal amount formed from the animal's body proteins.

Recently it has been shown by Sansum and Woodyatt[15] that the rise in glycosuria following ingestion of various toxic substances is not necessarily to be ascribed to sugar formation from these substances. The results obtained in the experiments to be discussed in this article are, however, not subject to this criticism, as only innocuous food substances usually representing normal constituents of animal diet were fed in this case.

In order to determine quantitatively the amount of glucose

arising in diabetic metabolism from a given protein on ingestion, it is also necessary to demonstrate that at least under the experimental conditions employed complete digestion of the material fed has taken place, together with absorption and elimination of the digestive products arising from it. In this regard a variety of physiological factors are now known to play a part. The researches of Mendel and co-workers have demonstrated that the rate and thoroughness of protein digestion is mainly dependent on the volume and texture of the material fed, as well as on the presence in the alimentary tract of other food and indigestible substances. It has also been shown that even the amount of water taken can markedly influence digestion.[16] When these factors are considered, however, digestion and absorption of proteins, whether of animal or vegetable origin, are with but few exceptions very similar.[17] These data were mostly unknown at the time when the chief part of the previous experimentation on glucose formation took place, and represent an uncontrolled source of error in many of these experiments. In our preliminary studies due consideration, however, was given these physical factors. The experiments have therefore been made throughout this entire series of investigations on fasting animals, and due care given to the quantity, volume and texture of the proteins fed. The water intake was also properly regulated. In a series of preliminary control experiments the hourly nitrogen and glucose elimination were also followed and exact time limits established which insured the excretion of these products arising from the ingested proteins. It was found possible in this way so to refine the technic that the glucose arising from proteins in the metabolism of the living animal could be determined with as great degree of accuracy as accompanies certain analytic procedures.

Inasmuch as the value of many recent diabetic researches, including the present studies, depends on how directly the experimental results obtained for phlorizin diabetes are applicable to human diabetes, a few remarks relative to this subject are not out of place. As is well known, various differences exist between these two forms of glycosuria. A detailed comparison of these conditions

cannot, however, be entered into the present article. The data bearing on this subject has been collected and ably discussed by Allen,[18] to whose monograph reference may be made. The writer's stand-point concerning the nature of phlorizin glycosuria may, however, be briefly stated as follows:

Phlorizin diabetes is produced by the action of the glucosid of this name and is regarded by many to be of renal origin, in sharp distinction to diabetes mellitus. This view, however, requires modification. According to Biedl and Kolisch[19] the liver forms glucose when perfused with fluid containing phlorizin. The writer[20] has observed that the glucose content of blood to which phlorizin has been added increases on perfusion through muscle. Underhill[21] has found considerable glucose formation to take place in phlorizinized animals in which the kidney function was eliminated. It has been fully demonstrated by Lusk in various ways that the body proteins are the source of sugar excreted by fasting phlorizinized dogs. From these and further facts which could be adduced the conclusion can be drawn that phlorizin affects the tissues of the body as a whole with sugar synthesis as a result. Phlorizin diabetes is therefore to be regarded just as is diabetes mellitus, a morbid process of general nature, characterized by glucose formation from protein.

The effect of phlorizin on the kidney has been the object of many investigations. The various data and arguments on this subject also cannot here be discussed. It may be but briefly emphasized that the importance of the kidney in phlorizin diabetes, though very great, has been overestimated, for neither sugar formation in this organ nor indeed in the blood can adequately account for the production of such large amounts of glucose as are excreted in this form of diabetes. The local action of phlorizin on the renal organs is, however, in all probability responsible for several of the chief differences observed between this and the human form of diabetes. The hypoglycemia and various phenomena connected with glycosuria and diuresis characteristic of phlorizin diabetes are thus best explainable as the result of the toxic effect of this glucosid on the renal function.

In further comparing these two forms of diabetes the lack of the clinical symptoms of diabetes mellitus in phlorizin glycosuria cannot be fairly regarded as representing a fundamental difference for the following reason: In judging of this side of the question it must be remembered that it is difficult properly to compare the reaction of a dog subjected for a short period of time to phlorizin injections with the eminently chronic manifestations of human diabetes. Many signs of human diabetes, however, are actually to be observed in phlorizinized dogs. Such are rapid loss of weight, polyuria, and at times polyphagia, thirst, delay in the healing of wounds, and tendency to local infections. Even cataract has been noted.[22] A further discussion of this important subject cannot be entered into here. There are good grounds, however, for accepting that phlorizin diabetes has many of the characteristics which would reasonably be ascribed to diabetes mellitus of the same intensity and duration, if such an acute form of human glycosuria were known. However, no attempt to establish the identity of these conditions can be reasonably made. On the other hand, sufficient evidence, it is believed, has been advanced to show that these morbid processes are much more closely related than is generally recognized.

Aside, however, from the general nature of these two expressions of diabetes, and especially in regard to glucose formation from protein, previous studies make it very probable that this process is essentially the same in both severe human and phlorizin diabetes. Body proteins break down in either case with the liberation of nitrogen and glucose in like amounts in both these conditions.[23] Glucose formation from ingested proteins has likewise been demonstrated to take place in phlorizin as well as human diabetes. The relation of the urinary glucose to the nitrogen, or glucose-nitrogen ratio, has been repeatedly recorded as practically the same for either condition when of maximum development.[24] A human case of phlorizin diabetes showed the same glucose-nitrogen ratio.[25] It is very probable that more similarities of like nature would have been brought out did human beings not present so many difficulties with regard to exact experimental study.

Granting then that the body proteins of diabetic man and the phlorizinized dog yield about the same amount of glucose in metabolism, does the same hold true for ingested proteins? We believe this is to be answered in the affirmative for the following reasons: It has been demonstrated in this laboratory[26] that very nearly the same amount of glucose was formed in phlorizinized dogs from the proteins of the living animal and from muscle protein, that is, from the chief bulk of body protein of either dog or man when fed to phlorizinized dogs. This work makes it very probable the glucose formation from proteins ingested by man is not likely to be different from that occurring in the dog, providing no fundamental differences exist in protein metabolism in general between these two species. This point is covered by the work of Osterberg and Wolf,[27] among others, who in elaborate studies could discover no salient differences between the canine and human in this respect. For these and other grounds we deem it justifiable to apply to the problems of diabetes mellitus the results of careful protein-feeding experiments made on dogs having phlorizin diabetes.

Glucose Formation form Isolated Proteins. After thus satisfying ourselves as to the reliability of the technic employed as well as to the value of the data obtained by this mode of experimentation, a representative group of chemically pure, isolated proteins was fed in a series of more than seventy metabolic experiments to diabetic dogs and the glucose yielded in the metabolism of the proteins ascertained. The proteins employed and the results obtained are shown in Table 1, which is taken from a preceding article.

TABLE 1.—GLUCOSE YIELDS OF INGESTED PROTEINS

	Casein.	Ovalbumin.	Serum albumin.	Gelatin.	Fibrin.	Edestin (hemp protein).	Gliadin (wheat protein).	Zein (corn protein).
Glucose yield in per cent.	48	54	55	65	53	65	80	53

This investigation clearly established the important fact that no difference in their sugar-producing capacity exists between animal and vegetable proteins, owing to their respective origins. The

amount of glucose yielded in metabolism could, however, be demonstrated to vary directly with the amount of glucogenetic amino-acids contained in each individual protein. Thus the wheat protein gliadin contains 43.7 per cent. glutamic acid, which is known to yield large amounts of glucose in metabolism. Gliadin yields the largest amount of glucose of all proteins hitherto examined. This is chiefly due to the large amount of this amino-acid present. Application of these new facts to the diabetic's dietary will be made in the latter part of this article.

Source of the Glucose Arising from Protein. At various times in the medical literature discussions have arisen as to what part of the protein molecule yielded sugar, and particularly as to whether the carbohydrate content of protein could be responsible for the metabolic glucose originating from this food class. This question can, in view of the experiments just recorded, be regarded as definitely settled in the negative. Through the studies of Lusk, Dakin and others[1] it has been conclusively demonstrated that the majority of the amino-acids entering into the constitution of proteins yield glucose in metabolism. Our own work has demonstrated the fact just alluded to, that the amount of sugar so formed from proteins is dependent on the amount of glucose-yielding amino-acids entering into the make-up of each particular protein.[13] Indeed, according to recent studies in the chemistry of proteins, carbohydrate as such is present in very insignificant amounts and is to be rather regarded as an impurity than a component part of the protein molecule. This fact has been borne out by our metabolic experiments in which pure ovalbumin was fed. This protein was formerly thought to contain considerable carbohydrate, but our results show that it yields actually less glucose than various other albuminous substances to which a carbohydrate group has never been ascribed. This is a point which seems as yet little known among authorities on diabetes. Thus von Nooden,[28] as late as 1912, believed that a carbohydrate group plays a role in glucose formation from protein.

Glucose Formation from Body Proteins; the Glucose-nitrogen Ratio. The origin of the glucose excreted in diabetes

has held the attention of investigators for generations. Proteins and fat are to be regarded as the chief possible sources of endogenous glucose excretion. In order to ascertain what amount of the glycosuria could be ascribed to protein and what to fat, the urinary nitrogen elimination has been studied in relation to that of sugar, that is, the glucose-nitrogen ratio. This has been done in order to determine the maximal amount of the sugar production from protein. Sugar excreted in excess of this maximum could be ascribed to fat.

But efforts to determine how much glucose can arise from protein through study of the urinary glucose-nitrogen ratio have met with many difficulties. Minkowski found that fasting depancreatinized dogs excreted glucose in relation to nitrogen as 2.8 to 1, and therefore calculated 45 per cent. as the maximal amount of body protein convertible into glucose. Lusk, using phlorizinized dogs, established the glucose-nitrogen ratio as 3.65 to 1, corresponding to a conversion of 58.5 per cent. of protein into sugar. Similar studies carried out on human diabetics have led to the vaguest results. Thus glucose-nitrogen ratios have been reported varying all the way from 0.01 to 1 to 12 to 1. Owing to this uncertainty most authors on diabetics attach at present very little weight to the importance of the glucose-nitrogen ratio.[28] With the aid of the improved technic already alluded to, the problem of the maximal formation of glucose from protein, however, could be definitely decided. With the same precautions as previously employed in the case of isolated proteins, muscle from various species of animals was fed to phlorizin diabetic dogs and the amount of glucose arising in metabolism ascertained as hitherto. By the aid of a new analytic method[29] developed for this purpose, it became possible accurately to determine the amount of protein present in the animal muscle used in these experiments and also to ascertain the nitrogen content of this protein. By this means we were enabled not only definitely to learn how much glucose arises from muscle proteins in metabolism, but what relation this glucose actually bears to the nitrogen of the protein,

that is, the real protein glucose-nitrogen ratio, which is not identical with the urinary glucose-nitrogen ratio. The protein glucose-nitrogen ratios for body proteins other than muscles were obtained in a similar manner and were found to average just about the same as in the case of the muscle proteins themselves. In this way the maximal formation of glucose from body proteins could be established by direct experiment. No great variations were found among various species, as Table 5, taken from our previous studies, demonstrates. The body proteins of man were thus found to yield 58 per cent. of glucose in the diabetic metabolism, corresponding to the glucose-nitrogen ratio of 3.6 to 1.

It follows from this fact that complete diabetes in man exists when the glucose-nitrogen ratio of the urine is about 3.4 to 1 (Janney and Blatherwick). This glucose-nitrogen quotient now established as a definite value consequently becomes of importance in the study of this disease, as it represents a definite index of its severity. The nearer this ratio as exhibited by fasting diabetics approaches the value 0 to 1 the better the prognosis which may be made. Conversely, the persistence of a high glucose-nitrogen ratio adds to the gravity with which the case is to be regarded, for not only is a maximal formation of glucose from the body proteins here taking place, but also oxidation of carbohydrates is impossible.

As a result of these studies it is evident that the urinary ratios higher than about 3.4 to 1 previously reported for human diabetics must be based on erroneous observation. These new investigations are also applicable to the question of sugar formation from fat in diabetes as previously alluded to. Owing to the demonstration that all the glucose and no more which can originate from protein appears in the urine of severe diabetic patients and of fully phlorizinized dogs during a fast, it is evident that fat is not to be regarded as an important source of urinary glucose in diabetes. This coincides with Mandel and Lusk's[30] observations that the feeding of large quantities of fat has no influence on the sugar output of phlorizinized dogs. Though from certain fatty acids

synthesis of glucose undoubtedly does occur, as has been demonstrated by Ringer and others, glucose formation from fat evidently plays no large part in the diabetic's economy. In the actual treatment of this disease it is known, however, that the addition of fats to the diet can lead to increased glycosuria and ketonuria.[31] This is, however, very probably to be regarded chiefly as a result of the stimulating action of fat on protein metabolism, for Lusk's[32] elaborate respiratory studies have demonstrated that certain intermediary products, which can also arise from fatty acids, act as a stimulus to metabolism. These facts are emphasized inasmuch as considerable doubt exists in regard to the question of sugar formation from fat.

Glucose Formation from Protein Foods (with the assistance of F. A. Csonka). *Glucose Formation from Meats, etc.* The investigations just described have rendered the solution of another very practical problem possible, that is, the exact determination of the amount of glucose produced from meats and other protein foods as a result of their metabolism in the diabetic organism. The detailed results of this study are reported in this communication. Various meats were fed to dogs made completely diabetic by the use of phlorizin and the amounts of glucose arising from the food ascertained under the employment of the same technic and methods as previously employed. These experiments are accordingly not described in full. The meat was prepared for analysis and feeding by removal of all indigestible portions, ground fine in a meat grinder, the amounts for each feeding weighed, and then stewed for one hour on a steam bath. This was done with the idea of obtaining results applicable to cooked food. The eggs for analysis and feeding were hard-boiled, and after removal of the shells, were passed through a meat-grinder, thoroughly mixed and moistened with water before feeding. In the meat for feeding, nitrogen, total solids and, for the sake of control, the total amount of substances reducing Fehling's solution, as well as the glycogen content, were determined with employment of the same analytic methods as previously. The results of these analyses appear in Table 2.

TABLE 2.—ANALYSIS OF MEAT USED IN FEEDING EXPERIMENTS, GRAMS PER 100 GM. MEAT*

	Total solids.	Nitrogen.	Substances reducing Fehling's solution.	Glycogen.
Lean beef (first two experiments)	25.35	3.32	0.18	Less than 0.1
Lean beef (last two experiments)	25.08	3.28	0.18	0.08
Chicken meat	25.54	3.7	0.29	Less than 0.1
Fish (halibut)	22.0	2.98	0.23	Less than 0.1
Chicken egg	26.9	2.1	0.31	Less than 0.1

The reducing substances and glycogen were present in too minute amounts to exert any obvious influence on the glucose yielded by the meat fed. This fact we have indeed demonstrated in another way. In earlier experiments muscle proteins were separated in considerable quantity from the other meat constituents and found in similar feeding experiments to give rise to precisely the same amount of glucose as did corresponding amounts of the same sample of meat previous to its being subjected to this procedure. Meat extract has also been demonstrated by Csonka[33] to be not glycogenetic, an important fact to be considered in diabetic dietetics. There is therefore no question but that the protein itself represents practically the entire source of the glucose derived from the meat. The meat was fed in very moderate quantities, chosen so as to administer the albuminous substances to the animals in similar amounts as our preliminary studies had demonstrated to be entirely assimilated and the products eliminated within nine hours. Meat is known to be very quickly digested, absorbed and its metabolites excreted by man and animals. Important recent studies of Mendel and Fine[34] have demonstrated that when fed in larger quantities than used in these experiments it is utilized to the extent of 100 per cent. by dogs. Lusk and his co-workers[9] have fed various amounts of meat to fasting phlorizinized dogs and demonstrated that even 500 gm. of meat is digested and eliminated as nitrogen and glucose within twelve hours after ingestion. As we have used much smaller amounts of meat and allowed twenty-four hours for elimination, there is no question as

* Analyses of rabbit meat have been published previously, Jour. Biol. Chem., 1915, xxii, 212.

to the completeness of digestion, absorption and elimination of the digestive products formed. This is also conclusively shown by the protocols of the experiments, in which, with the exception of chicken egg, the nitrogen and glucose of the ingested material is always seen to be eliminated within twenty-four hours.

The protocols of the feeding experiments accompany this article (Table 6). For the mode of calculation of that portion of the glucose excreted which is ascribable in origin to the proteins ingested Lusk's method, which is now generally accepted, was employed. Three or more separate experiments were performed in the case of each variety of protein food given. The results of the various experiments usually showed remarkably close agreement (see protocols). It was thus found possible to determine the extent of glucose formation from meat proteins with the same accuracy as that in the case of analytically pure proteins employed in earlier experiments.

TABLE 3.—GLUCOSE FORMED IN METABOLISM BY PROTEIN FOODS

Food.	From raw material, per cent.	From water-free material, per cent.
Beef	9.5	38
Rabbit	11.0	45
Fish (halibut)	12.0	45
Chicken	12.0	48
Chicken egg	9.5	36

In Table 3 are recorded the average results of the feeding experiments given in detail in the protocols. It is seen that the amounts of glucose produced from the meats of various origin show no more deviation than might be ascribed to experimental error. These results stand, therefore, in accord with data previously obtained in this laboratory, which showed that the body proteins of various species of animals all yield approximately like amounts of glucose in metabolism (Janney and Blatherwick).

The reliability of these results for the various meats studied we believe to be scarcely open to question, owing to the careful preliminary experiments alluded to. The same cannot, however, be maintained for the egg experiments, in which larger amounts of

material were fed than our later control experiments referred to above. Owing principally to this fact, in all likelihood a much longer time was required for the sugar and nitrogen arising from them to be excreted than in the case of the meats. The results obtained in this case are perhaps not so dependable as in the meat experiments. They are, however, very regular and do not vary more than the similarly obtained glucose values of the other protein foods. On these grounds they are included with this explanation in our report.

TABLE 4.—GLUCOSE FORMATION FROM PROTEIN FOOD; COMPARATIVE TABLE

	Water content, per cent.	Glucose yield, per cent.	Amount equivalent to 100 Gm. bread, Gm.	Calories per 100 Gm.
Beef, raw	74.8	9.5	642	150
Beef, broiled	54.0†	17.5	348	208
Beef, dried or smoked	54.3	21.0	290	185
Beef, canned or corned	51.8	18.2	335	270
Beef, roasted	48.2	19.5	313	241
Chicken meat, raw	74.5	12.0	508	197
Chicken meat, roasted	59.9	19.2	317	245
Rabbit, raw	74.7	11.0	555	...
Rabbit, broiled	61.4†	16.8	363	...
Halibut steak, raw	75.4	12.0	508	124
Halibut steak, fried	54.2†	22.3	255	173
Eggs, raw	73.7	10.3	592	153
Eggs, boiled	73.2	10.51	580	166
Eggs, fried	70.4†	11.6	526	160
Ovalbumin*	...	54.0	113	...
Gelatin*	...	65.0	94	366
Casein*	...	48.0	127	...
Corn protein, zein*	...	53.0	115	...
Wheat protein, gliadin*	...	80.0	76	...
Flour*	...	92.5		
Bread	34.0‡	61.0	...	277

The results of this series of protein food experiments, though obtained at the cost of considerable effort, are not directly applicable as they stand, however, to the problems of the diabetic's dietary. This is true as they are referable as calculated to raw material, except in the case of the egg feeding. By taking into

* Calculation based on water-free material.
† Writer's analysis.
‡ Analyses from Conn. Agric. Exper. Station Report, Sec. 1, Diabetic Foods, 1913.

consideration the loss of water in cooking, it is possible, however, to compute the amount of glucose which arises in the metabolism of such protein food as served. Such calculations have been made and appear in Table 4. As about one third of the water is usually lost in the culinary preparation, the glucose yields are correspondingly increased. It is apparent that a very considerable amount of glucose arises from meats. Owing chiefly to their higher water content raw, boiled or fried eggs produce but slightly more than one half the sugar which is formed from roast meat. Eggs are therefore to be regarded as a standard article of the diabetic's protein dietary. This experimental observation coincides with the experience of von Noorden with the human diabetic, as this clinician is accustomed to feed eggs in preference to other protein food in order to obtain a decrease in the glycosuria.

In the dietary tables for diabetics compiled by Carl von Noorden various foods are compared in regard to their carbohydrate content to white bread and equivalent values calculated. This method of comparison, as the present article demonstrates, is a rather inexact one, for the additional glucose formed within the organism from the proteins of the foods was not taken into account. In order then to supply a more proper basis for comparison of such foods from this stand-point it is necessary to ascertain the amount of sugar actually arising in the metabolism of bread. To accomplish this a final series of experiments was carried out, some of which have been reported in detail by Mr. Csonka[35] from another point of view. On account of its more constant constitution in these experiments, wheat flour was used in preference to bread. The same method of experimentation was employed as previously, with satisfactory results. It could thus be ascertained that flour produced an amount of glucose in metabolism equivalent to 92.5 per cent. of its water-free weight. From the data so obtained it is possible to calculate that the amount of glucose formed from bread in metabolism is about 61 per cent.

We were now finally in a position to calculate the amounts of protein food equivalent from the stand-point of actual glucose formation in the body to bread. These values appear in the last

column of Table 4. It is seen that about 3 to 3.5 parts of cooked meats correspond to one part wheat bread. In view of this fact it follows that rather more care in regard to the protein part of the diabetic's dietary should be exercised than is at present usually deemed necessary. In order to emphasize the marked glycogenetic property of proteins the bread equivalents of various food proteins have been added. Thus gelatin, recommended by von Noorden as a permissible food, produces 65 per cent. glucose within the body, and but 94 parts of gelatin are equivalent to 100 parts of bread. It must be remembered, however, that gelatin is eaten in the form of a jelly with a corresponding resulting reduction of the percentage metabolic glucose formed from it. It is nevertheless certainly no longer to be regarded as a proper food for diabetics.

With regard to the relative usefulness of the various kinds of bread in the diabetic's dietary, the following may here be observed. As nearly as can be at present approximated, the proteins of corn bread yield about 50 per cent. of glucose in metabolism, wheat and rye bread about 66 per cent. The results of the average analyses of Atwater and Bryant are, respectively, for graham (wheat), corn and rye bread, protein, 8.9, 7.9, 11.9 per cent.; carbohydrates, 52.1, 46.3, 35.9 per cent. From this and other data in this article the total percentile sugar yield of these important foodstuffs in metabolism may be estimated as follows: white wheat bread 61, graham wheat 55, corn 50 and rye 44 per cent. From these data it would seem as if rye bread should be preferred for diabetic use. Aside, however, from the fact that Americans as a rule do not relish rye bread, its caloric value is but three fourths that of the other varieties mentioned. Everything considered, then, it seems evident that white wheat bread is least desirable for the diabetic's use, corn bread and other cornmeal preparations most desirable; graham and rye bread occupying a middle position.

Glucose Formation from Proprietary Foods. So much emphasis has been laid by the physician on the necessity of excluding carbohydrates from the diet of the glycosuric patient that this has become the aim of the manufacturers in preparing diabetic foods. Indeed, it still remains the custom of physicians to

recommend at times such special breads and other products on account of their high protein and low carbohydrate content. Many falsifications and misleading statements in regard to the make-up of these foods have, however, been made on the part of their promoters. Analyses of such American commercial products by the state agricultural experiment stations, of German preparations by Magnus-Levy,[36] Janney[37] and others have already called sufficient attention to such misrepresentations. A study of the actual nutritive value of these preparations to the diabetic, however, is of more importance than the claims made for them commercially. It has been found possible to apply the experimental data previously obtained to these patent diabetic foods and thus to secure a knowledge of their real usefulness to the glycosuric patient. The results of this consideration are collected in Table 5.

The protein and carbohydrate values used in making these calculations are those compiled by the Connecticut Agricultural Experiment Station[38] from scientific investigations of these food products and are said to be regarded as reliable. The percentile amount of glucose produced in the body by these products (third column, Table 5) has been calculated by adding to their carbohydrate content the amount of glucose formed from the protein present, these latter values being taken from our experiments. It has been previously ascertained by the author that casein produces 48 per cent. of glucose. For plasmon and sanatogen, which are casein preparations, this value has accordingly been utilized.

TABLE 5.—GLUCOSE FORMATION FROM PATENT DIABETIC FOODS

	Protein, per cent.	Carbohydrate, per cent.	Glucose yield in metabolism, per cent	Amount equivalent to 100 Gm. bread, Gm.
Glidine, Menley & James, New York	91.4	1.0	66	92
Sanatogen, Bauer Chem. Co., Berlin	80.1	4.2	61	100
Plasmon, Plasmon Co., London	78.7	...	56	109
Diabetic Biscuit, Johnson Educ. Food Co., Boston	25.3	59.0	77	79
40 per cent. Gluten Biscuit, Kellogg Food Co., Mich.	35.8	54.0	79	77
80 per cent. Gluten Biscuit, Kellogg Food Co., Mich.	82.4	4.4	63	97

In order to ascertain the total amount of sugar derivable from gluten preparations it is necessary to ascertain the amount of glucose arising from the wheat proteins. This was done as follows: Of the wheat proteins about 90 per cent. are represented by glutelin and gliadin in the proportion of 10 to 8.4. These two proteins are the chief constituents of gluten. In the absence of actual feeding experiments the amount of metabolic glucose derived from glutelin was calculated as previously done in the case of other proteins, from the amounts of glycogenetic amino-acids obtained from it by hydrolysis.[27] The resulting value is 66.3 per cent. Gliadin produces 80 per cent. of glucose in metabolism, as determined experimentally. Accepting that the remaining proteins of the wheat gluten yield about 60 per cent. of glucose under the same circumstances, we can calculate from these data that the wheat gluten gives rise to about 70.5 per cent. of glucose in metabolism. This value has been made use of in compiling the table. It must be borne in mind that the values so calculated are only approximate, owing to the manner in which they are obtained. Thus the method commonly in use for estimating the amount of protein present in albuminous foods is quite inaccurate. Attention to this has been recently called by the writer.[39] Table 5, however, serves forcibly to illustrate the lack of value or indeed positive harmfulness of such commercial products when eaten by the diabetic. It may be remarked that sanatogen, plasmon and glidin, the last a German product identical to that appearing in Table 5, are recommended by von Noorden among foods permissible to all diabetic patients.[40]

A further important fact is also brought out by the data of Table 5. Contrary to the prevailing opinion, the use of food preparations with a high protein and a low carbohydrate content is of not much more advantage to the diabetic than that of others containing much more carbohydrate and less protein. This is well illustrated by comparing the Kellogg Food Company's 40 per cent. gluten biscuit with their 80 per cent. product. Although in the second case the protein has been increased from 35.8 to 82.4 per cent. and the carbohydrates decreased from 54 per cent. to

4.4 per cent. the glucose formed in the metabolism of this product could thereby be reduced from 77 to only 58 per cent. The chief lesson to be drawn from these considerations is obvious. Food products containing high amounts of proteins cannot be properly recommended for the use of diabetics, inasmuch as most of them present no advantages over wheat bread fed in like amount. As these articles are with few exceptions relatively very expensive, and, owing to advertising propaganda, the unsuspecting diabetic is often deluded as to their value, their sale should no longer be encouraged by either physician or layman. A comprehensive list of such diabetic foods has been published, with their protein and carbohydrate content, by the Connecticut Agricultural Experimental Station.[38] This may be referred to for determining the utility of diabetic foods other than the few examples quoted in this article.

Application of Results to the Diabetic Dietary. The experimental results described in this article were obtained with the aid of the same technic throughout. Optimal conditions for digestion and absorption were maintained for small amounts of protein foods in highly assimilable form which were fed to fasting subjects. Reasons have already been stated for regarding the glucose values as representing the maximal sugar formation from the protein material in each case. It is therefore justifiable to regard the tables given as a general index of the utility of proteins and protein food for the diabetic and to draw comparative conclusions from these data as has been done in the foregoing pages.

In actual practice, however, such ideal conditions as the experimental do not exist. The various factors influencing the extent and rate of protein assimilation here become of decided consequence. Among them are the texture of the food served, thoroughness of mastication, presence of substances other than protein in the diet, volume of indigestible material in the intestinal tract, variations in the tolerance to carbohydrates, the effect of exercise and psychic influences. On applying, then, to the diabetic's dietary the results of the researches described in this article, the

influence exerted by these factors must be regarded. A very palatable and digestible protein food, though shown to yield much more glucose by our experiments than another example, may possibly, through poorer digestibility and absorption, or the fact that these processes take place more slowly in this case, be the cause of less sugar excretion than second food on their administration to patients. Von Noorden's view that vegetable proteins more favorably influence the glycosuria than does meat, though based on insufficient experimental proof, is, however, susceptible of explanation on these grounds. Vegetable proteins, as we have seen, actually form as much, and in some instances more, glucose in metabolism than do meats, when both these forms of food are fed under optimal conditions. If, however, vegetables are for any reason eaten by diabetics in a form less or more slowly assimilable than a given amount of meat, glucose formation would be correspondingly decreased in the former case. Further exact experiments seem here indicated.

The aim of modern cooking, however, is to render all food, whether animal or vegetable, highly palatable and digestible. The alimentary factors alluded to play not so great a role in better practice as might be supposed. With some possible exceptions, these variables would, especially in better households, tend to exert a more similar influence on the course of protein digestion. For this reason the glucose values of the tables in this article are believed to be applicable to the human dietary in a comparative way, even though they may not always represent the precise extent of glucose formation under the special conditions prevailing at the time of their ingestion. Thus, protein foods *A* and *B* may be equivalent to 80 and 60 gm. of bread, respectively, as experimentally determined. Being eaten under mutually the same but less favorable conditions by a diabetic, each protein might undergo 90 per cent. absorption. Therefore the values in the tables still remain criteria of the relative utility of these proteins. It is to be emphasized, however, that the results of the series of experiments here reported are to be regarded merely as a general gauge of the relative adaptability of protein foods to the diabetic dietary.

The Choice of Diet for Diabetics. The experimental data described in this communication emphasize the fact that large quantities of glucose are formed within the diabetic organism from food proteins. Evidently the classic severe strict diet, consisting chiefly of protein, still represents large quantities of sugar-forming material, however carefully carbohydrates are excluded from the articles consumed in such food. It seems, in view of these new results, scarcely a wise procedure to strive to eliminate every gram of carbohydrate from such a diet, inasmuch as nearly two thirds of the proteins fed are found to be converted into glucose in course of metabolism by the diabetic. As it is a matter of clinical experience that large quantities of fats serve to increase both glycosuria and ketonuria, it may be fairly asked what then remains for the diabetic to eat if protein, carbohydrates and fats all contribute to increase the amount of sugar lost to the body in the urine.

This question has indeed been logically answered by Allen, whose well-known treatment has emphasized the good results to be obtained from a complete fast. The rationale of the Allen treatment becomes more evident when one is mindful of the fact that not carbohydrates alone, but all the three great classes of foodstuffs may give rise to increased glucose formation. Thus it becomes apparent that only by total exclusion of all food, a complete rest can be given to the sugar-utilizing function of the organism.

The diabetic, however, cannot refrain indefinitely from food. How then feed him? In view of the series of experiments here reported it is likely that a diet containing moderate amounts of protein and fat and low amounts of carbohydrate is after all the most judicious one to be employed. It seems that only by very discriminatingly balancing the various advantages and disadvantages of each kind of foodstuff can the proper quantity for a given case be best determined. With the use of the food table giving the equivalents of meats and bread, the amount of protein food in relation to carbohydrates can be easily calculated.

TABLE 6.—PROTOCOLS OF FEEDING EXPERIMENTS*

Protein food administered.		Nitrogen fed, gm.	Weight of dog, gm.	Period hr.	Urinary nitrogen, gm.	Urinary glucose, gm.	G:N	Extra glucose.	
Kind.	Amt., gm.							Amt., gm.	In terms of material fed, per cent.
			9.95	24	9.23	32.64	3.54		
				24	9.12	34.9	3.83		
Lean beef .	50.0	1.66	....	24	8.64	29.1	3.37	4.46	8.92
				24	6.87	22.22	3.23		
			7.0	24	8.58	31.8	3.7		
				24	5.87	20.7	3.53		
Lean beef .	35.0	1.16	....	24	8.19	25.6	3.13	3.3	9.43
				24	8.18	26.79	3.28		
Lean beef .	89.6	2.84	8.6	24	8.98	28.17	3.13	8.34	9.30
				24	7.38	23.57	3.19		
				24	5.77	18.64	3.23		
				24	5.23	16.85	3.24		
				24	4.84	16.26	3.36		
				24	4.76	14.24	2.99		
Lean beef .	108.02	3.5	7.0	24	6.79	20.39	3.01	9.57	8.86
				24	4.2	13.86	3.3		
				24	8.03	29.23	3.64		
Lean beef .	91.67	2.97	9.9	24	9.88	34.63	3.47	9.89	10.79
				24	7.71	27.42	3.55		
				12	5.12	16.78	3.28		
				12	4.77	14.52	3.05		
Chicken meat	134.85	5.0	10.0	24	10.89	37.78	3.47	19.14	14.2
				24	7.76	24.75	3.19		
Chicken meat	128.1	4.75	9.1	24	9.19	28.25	3.07	14.23	11.1
				12	3.89	15.27	3.92		
				12	5.74	19.31	3.37		
Chicken meat	82.93	3.07	6.1	24	8.44	28.73	3.4	10.91	11.86
				24	5.57	18.18	3.27		
Chicken meat	74.16	2.75	5.9	24	6.27	21 43	3.42	9.48	12.79
				24	4.4	15.55	3.53		
Chicken meat	74.16	2.75	5.5	24	5.98	19.12	3.2	7.98	10.76
				24	3.69	12.5	3.39		
				12	4.77	16.26	3.41		
Halibut .	167.8	5.0	10.0	12	4.16	14.03	3.37		
				24	11.23	37.04	3.3	15.92	9.49
				24	2.67	8.03	3.01		
				24	10.26	31.71	3.09	13.64	9.54
Halibut .	143.1	4.25	8.5	24	7.19	21.58	3.0		
				24	3.49	10.92	3.13		
				24	4.73	15.43	3.26	6.6	10.33
Halibut .	63.97	1.9	3.8	24	3 75	11.56	3.09		
				12	4.41	16.95	3.85		
				12	3.92	15.75	4.02		
Chicken egg	176.2	3.7	7.4	24	10.5	40.28	3.83	16.04	9.11
				12	3.95	16.95	4.29		
				12	4.31	16.05	3.73		
				24	7.82	27.55	3.52		
Chicken egg	154.8	3.25	6.5	24	8.44	31.25	3.7	10.1	9.11
				12	3.31	12.66	3.85		
				12	3.07	10.75	3.5		
				24	12.49	40.82	3.3		
				12	5.53	21.05	3.8		
Chicken egg	390.4	8.2	16.4	24	15.6	61.21	3.93	40.76	10.44
				12	4.8	20.13	4.28		

* For protocols of rabbit experiments see Janney, N. W., and Csonka, F. A.: Jour. Biol. Chem., 1915, xxii, 203.

SUMMARY. A critical study of diabetes mellitus and phlorizin diabetes has led to the conclusion that glucose formation from protein in both these conditions is essentially the same. It is therefore justifiable to apply the very much more accurate results which can be obtainable in phlorizin experiments to the study of human diabetes. By employing a carefully controlled technic it was found possible to quantitatively determine the amount of glucose formed in the organism from ingested proteins. The chief results of previous investigations of the writer and co-workers are collected. They are as follows: Isolated proteins were found to yield large amounts of glucose in metabolism, varying from 48 to 80 per cent., according to the protein examined. Contrary to existing opinions, the animal or vegetable origin of proteins bears no relationship to their ability to produce glucose in the animal organism, this function being found to be mainly dependent on the amounts of sugar-yielding amino-acids entering into the constitution of these various proteins.

The formation of glucose from body proteins was also studied. It could be shown that body proteins of man and animals yield about 58 per cent. of glucose in metabolism. The nitrogen of these proteins bears about the relation of 3.6 to 1 to the glucose formed from them. This definite establishment of the glucose-nitrogen ratio is of value in the prognosis of diabetes. Cases showing such a high urinary glucose-nitrogen ratio, averaging 3.4 to 1, are to be regarded as grave. The lower the ratio the more favorable is the prognosis. As the glucose excreted by the fasting diabetic is of protein origin, sugar formation from fat does not take place to any great extent in this disease.

New experiments relative to glucose formation from protein foods are also reported. The amount of glucose originating in the diabetic metabolism from various meats was ascertained with use of the same technic as hitherto. In von Noorden's food tables for diabetics, glucose formation from protein has not been taken into account. By adding the amounts of glucose yielded in metabolism by the proteins of a given food to its carbohydrate content it is possible, however, to ascertain the actual amount of sugar, both

set free and formed in the metabolism of such foods. A more accurate table could thus be constructed showing the relative adaptability of protein foods to the diabetic dietary, as compared to equivalent amounts of bread. Finally, various proprietary protein foods were studied in like manner. It was found that such preparations present no advantages over equal amounts of bread when fed to diabetics, as the large amount of protein present leads to the formation of considerable glucose in metabolism.

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DISCUSSION

Dr. James E. Talley: We are greatly indebted to Dr. Janney for this most interesting and helpful paper. It is gratifying to have a scientific demonstration of the fact that the protein of egg is better borne than other proteins. Simply empirically some of us had come to this conclusion from our experience with patients who had poor tolerance some time after their starvation cure. Satisfying calory needs, the appetite and the family apprehension with these patients is a troublesome problem.

Dr. George M. Piersol: I have nothing further to add, except that it is interesting to have more scientific data concerning the rationale of the Allen treatment, which we all agree in the last year has proved to be the most successful means of combating diabetes. It has always been a matter of conjecture and of some controversy why it has worked as it does. All evidence tending to explain this is of great interest and value.

Dr. Janney (closing): I scarcely think there is much to add, except that I would like to sound a warning, not to take these results too positively. They are, as I tried to emphasize, to be considered, if perhaps the most reliable known at present, still merely as a general guide in the protein feeding of diabetics. To accept that a certain protein should be entirely discarded for another, owing to a difference of a few grams in sugar formation, is going too far, owing to the various digestive and other factors which play a role. When, however, the glucogenetic capacity of one food is strikingly less than that of another (for example, eggs as compared to meats, or meat as compared to commercial preparations high in protein), then it is that the practical utility of this new data becomes most important.

THE ADVANTAGES OF MILITARY TRAINING FOR YOUNG MEN AND THE PHYSICAL CULTURAL VALUE OF THE PREPAREDNESS MOVEMENT[1]

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The idea of general military training, even of compulsory military service, in this country is not a new one. In a letter written from Monticello, dated July 18, 1813, Thomas Jefferson, ex-President, writing to James Monroe, then Secretary of State in the cabinet of President Madison, in a reply to a letter from the latter, uses language which shows that both of these distinguished American statesmen foresaw the necessity of and advocated both universal military training and compulsory military service. Mr. Jefferson says, referring to Monroe's letter:

"Every line of it is replete with wisdom, and proves the necessity of obliging every citizen to be a soldier. This was the case with the Greeks and Romans, and must be that of every free State. We must train and classify the whole of our male citizens and make military instruction a regular part of collegiate education. We can never be safe until this is done."

The title of the paper I was asked to prepare is "The Physical Cultural Value of the Preparedness Movement." To tell the truth, the "preparedness movement" is still in the discussion stage, and includes many more or less futile and some very absurd schemes for making ready for national defense. Any effort in this direction is being vigorously opposed by many determined and loquacious orators. Under these conditions the only physical

[1] Read June 7, 1916.

cultural value of the movement is the increase by exercise of the lung power of the contestants.

If, however, by "preparedness movement" is meant the enforcement of general military training for our young men, which many men well qualified to judge consider the only efficient means of providing an adequate force of trained men for national defense, I am convinced that in time such a movement would result in a very material physical improvement of the men of our country. Assuming then that this is what was intended, I have given this necessarily short, and I am afraid desultory, paper the title of "Advantages of Military Training, Generally Applied to Young Men," and these advantages are by no means confined to improvement in physique, important as that is, for there are many other things a young man gets out of military training which I shall speak of even though they may seem not directly connected with the main subject.

In the many and voluminous articles we read these days on preparedness, the many more or less futile schemes that have been proposed to enable the country to elaborate a military reserve to be called out in case of necessity, the fact that systematic military training for growing young men is an excellent thing in itself, a very valuable part of the education of a boy, seems to have been lost sight of. There can be no doubt that there are many and great advantages to be gained by putting all young men, who are not mentally or physically disqualified, through a course of military training, advantages I mean to the young men themselves, even if they were never called on to do duty as soldiers in the field, though, of course, it would be of enormous value to the country to have within call a large body of potential soldiers.

Compulsory general military training, so dreaded and so denounced, and so often characterized by that most frightful of adjectives "un-American," would be, I believe, an excellent thing for our young men, and a very valuable part of their education; would make them better men, physically, mentally, and morally; better citizens; more efficient workmen, business men, and professional men.

We live in a veritable riot of individualism. The average American recognizes no duty to the State, and the average boy is brought up to believe that making money without working for it, keeping out of jail, and consulting his own comfort and inclinations are the only things worth considering.

Observe a gathering of young Americans, hear them talk, and notice their frequently poor physique, their cigarette-stained fingers, their loose-jointed, "slack-twisted" appearance, their awkward gait; go to a base-ball game and sit among the bleachers and note the appearance and manners of the young men and boys you see. You will not come away favorably impressed with young American manhood. And yet it is good material—excellent stuff out of which good men can be made. Inculcate into these young men some sense of duty to the State, some respect for constituted authority, based not on fear of the police but on reason and a realization of its necessity for the common good, feed them properly, give them systematic physical training, teach them discipline, self-control; in short give them a period of military training with all that it implies. Can you doubt that it would make them all-round better men? Can you think of any better method of educating them for useful citizenship? Compare the graduating classes at West Point, Annapolis, or the Virginia Military Institute with the classes at colleges, where, if there is any military drill and training it is desultory, not a matter of much interest to the faculty, and is gone through more or less perfunctorily. The difference is, I assure you, very manifest.

Athletics as practised at schools, colleges, and universities does not produce large numbers of symmetrically trained men. The tendency is to select a few, train them intensively in certain athletic specialties, often overtraining them to their permanent physical detriment, while the great mass of students exercise only their lungs in inciting their selected athletes to further efforts.

It may seem irrelevant to discuss advantages other than physical that result from military training, but it may be safely asserted that they are many and that they form a very valuable part of the education of a citizen. I doubt if there are in any civilized

country in the world, as in ours, so many young men who grow up in a spirit of practical lawlessness; who have no idea of obedience, respect for constituted authority, or the most rudimentary conception of what is called discipline. They know or care nothing of the value of team work, the enormously increased efficiency of a mass of trained men working smoothly together to accomplish a definite object, over the same number of men just as willing and anxious to do good work, but unaccustomed to working under discipline, without system, each man acting on his own initiative. This can best be taught by a period of military training, and makes the difference between an army and an armed mob.

The value of this kind of training was well illustrated after the earthquake and fire in San Francisco in 1906, and later in the disastrous floods in the middle west, and on many other occasions when the rescue work was non-military, but when small bodies of trained soldiers proved much more effective than much larger bodies of untrained men, no matter how strong and willing the latter might be. In Mexico today the civilian truck-drivers managing motor trucks and used to dealing with untrained men, express the greatest surprise at and admiration for the efficiency of the trained soldier, when called on to assist them, as compared with civilian help. Will not the average young man, who has undergone say a year of this kind of training, be a better workman, farmer, lawyer, architect or doctor, for the experience? I firmly believe he will. He will be a man of better physique for it; he will have learned something of personal hygiene, developed habits of cleanliness, order, system, punctuality, as well as the habit of instinctively subordinating his own inclinations to the general welfare. He will have led an open-air life, have been judiciously exercised, given abundant simple but properly selected and prepared food, carefully calculated to produce the maximum benefit. The average American family is not judiciously fed, and in this one matter of diet alone the military training will be of value in building up the young man in barracks or camp. The average American kitchen is more notable for waste of material than for good cooking. There was a vague idea when our troops

occupied the Philippines that the natives had by long experience reached the ideal diet for the tropics, and that rice, fruit, and fish, on which the lower class Filipino largely subsists, made up the proper ration for hot climates. The fact was that the natives lived on this diet because it was all they could easily get. When they enlisted as scouts and were given a much more generous diet (to which they took very kindly indeed) combined with exercise, they improved rapidly and very markedly in weight, muscular strength, and endurance, and became physically much more vigorous men than their brothers who did not enlist.

Can there be any doubt that such a well-balanced diet as the soldier's ration affords, combined with out-door life, active exercise carefully calculated to develop the entire body, and not carried to excess, would be of enormous benefit to the growing young man?

If you could see, as I have in many thousand cases, the round-shouldered, shambling, awkward recruit brace up, and in a few weeks of setting-up exercises, become a straight, up-standing, alert young soldier, you would, I am sure, agree with me that it was well worth while. The young man has by that time developed a certain degree of self-respect, he has had aroused in him the spirit of emulation and a desire to present as good an appearance as the best of his comrades. Incidentally he has learned what I believe is an excellent lesson for the average young man—not to take himself too seriously: that is, he has acquired a sense of proportion, a realization of the relative value and importance of people and things—a very valuable item in the education of any man.

Undue conceit is not popular and cannot long survive in the barracks, and, if the recruit is inclined to overestimate his own qualifications or exaggerate his own importance, he soon reaches his proper level under the not too subtle badinage of the barracks. A little ridicule is a potent factor in bringing young men into line, and it has a healthy influence in the life of the young soldier. I have seen this method applied in a large private school for boys with excellent effect in enforcing discipline.

But we are now principally concerned with the beneficial effect

of military training on the physique of young men. To get the best effects we should get very young men, preferably from eighteen to twenty, but preliminary physical training should be begun even earlier, say at fourteen.

The Boy Scout movement has demonstrated very well the excellent effect of even desultory physical training on young boys. The quasi-military features of this organization attracts the boys; the "gang spirit," present in all normal boys, is turned into useful channels and utilized as team work. The hikes and camping parties combine a pleasant, interesting occupation with instruction of a very valuable kind of out-door exercises, and the boys develop physically under conditions that to many of them would be unattainable except for the Boy Scout movement. It is certainly a step in the right direction.

One of the evidences of physical improvement as the result of military training that it is difficult to measure or prove by statistics but which is nevertheless an actual and important result, is the increased resistance to the inroads of infectious diseases that is developed by judicious military training. I can illustrate this by my own experience during the Spanish-American War. I was in command of a hospital ship and was ordered to Santiago, Havana, and Porto Rican ports, and Manila, to bring home some of the débris of the forces engaged in those islands. The principal morbidity was from typhoid fever, and we carried a great many cases, and many of them died at sea. Those who succumbed were almost invariably volunteers or very raw recruits for the regular army, while practically all the older soldiers recovered. This was so well recognized by the surgeons on duty in the wards that a bad prognosis was given whenever it was found that the patient had had little or no military training. The unfortunate lads, enlisted only a few weeks before, when there had been no time for physical training, had much less resistance than the better seasoned soldiers. That, too, explains the very high mortality rate in the volunteer camps in the United States. If these camps had been occupied by trained soldiers, used to taking care of themselves in camp, hardened by physical training, the mortality would certainly have been lower, probably very much lower.

The idea of compulsory general military training seems to be abhorrent to the American and English people, but does it not seem that the State has as much right to demand of its citizens protection from invasion as to call on them to pay taxes to defray the government expenses? No one is asked to pay taxes. He is forced to do it. He is not asked if he wants to, for the answer to that question would be an emphatic "No!" But we are again straying from the subject of the physical improvement to be expected from military training. So many things are of interest in this connection that it is difficult to keep to the beaten track.

The subject of the physical development under systematic training of the recruit has been studied by medical officers of the armies of all nations, and the same training that produces such good results in the more mature recruits should do even more for younger men, who are still growing and who have not yet reached a stationary period of physical development. Of course there must be a firm foundation on which to build, and careful physical examination must be made to exclude young men who are permanently disabled mentally or physically, but it is surprising to see the rapid improvement under systematic training and good feeding of men classed as "under weight for height," "poorly nourished," "poor muscular development," "anemic," etc., but who show no evidence of serious definite organic disease, or who are not crippled.

Previous hard manual labor is by no means an equivalent for judicious physical training. It may develop one set of muscles at the expense of others, and it may take time and special gymnastic exercises to eliminate the muscular asymmetries produced by some occupations. Under training the man becomes straighter, chest capacity increases, he gains weight, and his muscles increase in volume and improve in tone. Or, if there is a tendency to obesity, the fat disappears and the whole physique improves. Rossignol, writing of recruits for the French army, says: "Young men whom one would consider too weak to endure the hardships incident to military life acquire after six months' military training a general development of the body we should scarcely be bold enough to hope for, and become, under the influence of exercise, strong and robust soldiers." When physical training is properly

carried out, men are educated to act with cohesion, rapidity, and accuracy, each soldier having his individualism developed, and his intelligence sharpened; individual serviceability is increased. A late superintendent of West Point reports that the physical tests and measurements of cadets showed very marked gains at the end of the first year; gains in weight, chest capacity, and muscular measurements, and also in practical tests of strength on the horizontal and parallel bars and broad jump.

Medical Director Beyer, of the Navy, made a series of observations on the cadets at the Naval Academy. The system of records at the Naval Academy made it possible to compare the physical development of the cadets before and after the establishment of systematic physical training. He found, as the result of this training, a very marked increase in lung capacity, and even in the height of the cadets, but, as these boys averaged only eighteen and a half years of age, the increase in height, as they had not all ceased growing naturally, can scarcely be attributed to the training. In other respects, however, the improvement in physique was very marked after the establishment of systematic training.

According to an English medical officer, out of 15,504 British soldiers, who completed a course of three months' physical training in 1887 in the military gymnasia of the United Kingdom, the average increase was in weight, $2\frac{1}{2}$ pounds; chest measurement, $1\frac{1}{4}$ inches; forearm, $\frac{1}{2}$ inch; upper arm, $1\frac{1}{2}$ inch; thus accomplishing in three months a very marked physical improvement. And these soldiers were all mature men. Even better results can be expected if the training is given younger men or boys. At one time 12 non-commissioned officers of the British Army, selected from infantry, cavalry, and artillery, were sent to McLaren, an English army surgeon who made a study of physical development. These men ranged from nineteen to twenty-nine years of age, 65 to 72 inches in height, and from 128 to 172 pounds in weight. As the result of three months' training he says: "The muscular additions to arms and shoulders were so great that by the fourth month the men could not wear the tunics that had fitted them easily before, and new clothing had to be issued to them. One of

these men gained 4 inches in chest measurement. All increased in weight, the average being 10 pounds gain. One gained 16 pounds in less than four months, and this gain was not in fat, but in muscle increase." McLaren adds: "But the greatest of all changes, which cannot be recorded in figures, was the great improvement in bodily activity, dexterity, mental alertness, and endurance to fatigue, a change a hundred times more impressive than any that can be shown by tape line or weighing chain." This is a fair illustration of what can be accomplished in a very short time by judicious physical training in men averaging twenty-four years of age. With younger men and longer training it will be, as it always has been, even better.

I have, in thirty-five years' experience in the army, seen many thousand young men develop under military training from round-shouldered, untidy, shambling, awkward boys with flabby muscles and pasty complexions, into well set-up, clean, alert, active young soldiers, after a few months of setting-up exercises and squad and company drill. Six months of this training can work physical wonders on rather unpromising material. As Colonel Woodhull, of our own service, expresses it: "As a consequence of efficient military hygiene, which involves mental and moral as well as physical training, the average recruit is a far better man at the end of his term of enlistment than he was at the beginning, if there is any discipline worthy of the name in his company. He has learned obedience, promptness, responsibility, order, the value of co-operation, the importance of cleanliness and regular habits, and proper physical exercise, all important elements in the education of any young man."

Another faculty developed by the associations of military life is physical courage, or at least the excellent imitation of it that is engendered by a frame of mind in which a man is much more afraid of his comrades' contempt than of the enemy's fire. A spirit of manly resolution, cheerful endurance of physical discomforts and even hardships are certainly most important attributes for any man, and these are part of the unconsciously acquired qualities of a soldier, and develop *pari passu* with his physical improvement under military conditions and training. Another faculty he

acquires is promptness in deciding the proper action to take in time of emergency.

I believe then that a systematic course of military training throughout the country, widely applied and given practically all healthy young American men, would result not only in the general physical improvement of the race, but in very valuable character building.

Time will not permit, nor is it necessary to go into minute details of the actual exercises of the recruit, but the results have amply justified the time, study, and effort spent on them, and the military surgeons of all civilized nations agree as to its great value. Military training, generally applied, has made of the Germans most efficient workmen, mechanics, business and professional men, and the Swiss training has resulted in the development of one of the most vigorous and generally efficient races of Europe. I do not believe that, leaving out of consideration entirely its value from a military stand-point, any better method for the physical improvement of the race could be devised than to require a year of military training of all able-bodied adolescent American men, and as a part of their education it would be of inestimable value. The time could not be more profitably spent. It would start them in life with a knowledge of, an instinct for, personal hygiene; physical, mental, and moral training; and would be a means of inculcating habits of life and modes of thought and action that would help to make them strong, self-reliant, and efficient citizens. Even if we were absolutely secure from international complications, and should never need a soldier in the uniform of the United States, general military training would do much, I believe, toward the physical and general betterment of the race. As, unfortunately, we are not entirely secure in our isolation, and may be said to live in a fool's paradise, located on the edge of a very active volcano, we would kill two birds with one stone by making our young men more efficient citizens, improving the average physique and general efficiency of the race, and at the same time provide a large mass of men trained to some extent at least to be of value for national defense.

As the result of military training young men return to their

homes with strong and healthy bodies. Statistics show that their children are healthier than those of men who have not had training.

They take with them habits of orderliness, cleanliness, precision, alertness of mind, and concentration of attention. They acquire the habit of discipline. When they return to civil life these habits are fixed parts of their character.

A discharge from the service with character "excellent" is a very valuable recommendation for a man, and will in many places secure him prompt and lucrative employment.

One of our national weaknesses is that we are all too much given to consulting our own individual comfort, interests, and convenience. We are all like Artemus Ward, who was perfectly willing to sacrifice his wife's relations on his country's altar.

Nobody wants his or her son to be taken away from school or from some lucrative occupation and sent into barracks or camp for a year, but he or she is quite indignant that the neighbors do not see their plain duty and send their badly brought-up sons to spend a year in military training.

If the American people could only be brought to a realization of the fact that they owe something, a great deal, to their country, that their private wishes and inclinations must give way to the interests of the State, the general welfare, something might be accomplished, but anything an American is called on to do that he doesn't want to do is an invasion of his sacred liberty and arouses at once fierce opposition. How long will this liberty last if no one is prepared to defend it, and what better preparation can be made than to train up our young men to be able to do their part when trouble comes, incidentally giving them a most valuable physical, mental, and moral education that will make them sounder, stronger, more efficient citizens, even if, as we all fervently pray, the day of wrath is indefinitely postponed? It is a safe and cheap form of insurance in which the policy-holder is also the beneficiary; a cheap and valuable method of education, that would result in a very material betterment of the race, at the same time securing a military reserve of incalculable value, if it is ever needed, and what man can say when that may be?

INDIVIDUALISM AND DECADENCE[1]

By ROBERT T. MORRIS, M.D.
NEW YORK

In taking up the question of struggle, we must begin somewhere; let us begin no farther back than that of the struggle of inorganic elements for position in the periodic table. You may begin with the struggle of the earth with the ether if you please, but let us begin with the struggle of inorganic elements for place in the periodic table. A colloid organism, the cell, next finds itself promptly in conflict with some microbe. A man is nothing but an aggregation of single-celled amebæ. These amebæ are carrying on their contest not only with bacteria, but individual cells are engaged in struggle with each other. According to the laws of continuity, struggle occurs, not only between the cells of an individual, but between individualism a family, between families in a town and between towns in a State, between States in a great organization of States. That conflict is carried on continuously and will continue in the future as in the past because it belongs to the laws of evolution. If we cared not who governed us, we might, like Quakers, avoid warfare for a long time. As civilization advances the periods of peace between large civilized groups of people will be of longer duration, but the warfare and the struggle more terrible when it comes.

Warfare by arms, whenever it does occur between nations, will be brought about by politicians, because of a basic feature in human psychology relating to groups. When groups of people are formed as nations, certain individuals feel an instinctive responsibility toward protection of the whole group. Methods in government are then formulated. Next in order comes struggle between individual diplomats and individual politicians for

[1] Read June 7, 1916.

supremacy of individual ideas in relation to protection of the nation group. Then the weaknesses in human character appear among the men who are in control of the destinies of a nation. Biologists are not prone to believe that the Kaiser willingly precipitated the present European war. His life history has been one of tremendous constructive effort, and a stupendous destructive effort would belong more naturally along lines of known psychology to politicians with selfish ends in view who were enabled to overset the will of a previously dominant constructive individual. (Principles relating to the fundamental features of warfare may be found at the library in a recently published book entitled *Microbes and Men*.)

At the present time almost all the Aryan nation groups are declining but the progressing Slavic groups are bound according to the laws of Nature to attack the rest of us at some time in the not very distant future. Then again a nation like Japan which has formerly been content with esoteric philosophy will struggle for dominion over the world whenever such a nation develops exoterically.

Nations of dominating force are undoubtedly to come out of the Orient from time to time, when they can disentangle themselves from esoteric philosophy. The motives for warfare will depend upon food supply questions basically, and these will include lesser economic motives, which may in turn be influenced by religious feeling or by desire for revenge. We must remember that during the next couple of aeons hundreds of wars will naturally be fought upon the very ground upon which we are standing this evening. Some of these wars will be between people of kinds of which we know nothing in this century. Mass action of the people in a nation of the future, as in nations of the past will be accomplished through the agency of patriotism. Patriotism is a nasty little prejudice, given to man by nature apparently for the purpose of keeping him in herd form, in nations, and this prejudice is flatly opposed to the beautiful ideal of brotherhood of man. The cynic says that the brotherhood ideal is more readily maintained on a large scale when large business interests make it desirable

for the bankers to be the priests. It is true that struggle by force of arms is more frequent between groups of people who are poor than it is between groups of people who are rich. Various tribes among the American Indians were incessantly engaged in trying to do to each other what the white man did to them more thoroughly. The same thing is going on in Asia and in Africa. A dozen wars of more or less interest were going on in different parts of the world when the European war began in 1914. If the sound of all wars could be brought within one hearing distance it would make a continuous hum like the monotonous droning of a great wheel in the shop century after century, without a moment of cessation.

We have without doubt a need for military training in this country, provided we have a sentimental wish to retain our identity as a nation. If we do not care to retain our identity as a nation, then we may go on in the same way as we are now going, developing that individualism that leads to early decline and which makes us more vulnerable to the attack of a predatory nation, and the predatory nations are developing on all sides.

Our United States of America is at present a big, helpless, fat, juicy rabbit waiting to be taken. We are too fat to fight.

Let us consider the elements with which we have to deal. The Puritan came here because of ideals in religion, because of ideals in character, because of ideals in intellect. The Dutch came here for good solid economic reasons. The cavalier came here in order to make a profit, and along with the cavalier movement came a very large number of criminals. It was believed that this new country offered a better field for criminals than did the old sapient field in Europe.

Now, the Puritan, Dutch and cavalier, left to their own resources in this country, found that it was necessary to combine for protective action, and the undesirable elements were disposed of in a natural way. The mass purified itself and we had a very fine foundation in government and in administration.

Then came another and greater influx of people from all parts of the world, people with artistic ideas, people with anarchistic

ideas, and what is more significant, people with traditions, traditions belonging to their individual nations. We have the Irishman, hearty, poetic, wasteful, and who would prefer to exchange a fact for a fancy; who fights only with his fists, and walks next week arm in arm with the man who knocked him down. We have the Italian who fights with the stiletto, who carries a life-long grudge, and who introduces the mafia. We have sombre Scandinavian and the fun-loving Frenchman. We have the German trained in efficiency until it has become his religion. We have the Russian Jew with his average birth-rate much higher than that of any other race under urban conditions. All the different elements are being hybridized. We are having crosses between species, and this does not give large possibilities for mass action.

Plant breeders and animal breeders know that specific hybrids, or crosses between species do not make durable types. The strongest nations are those which belong to crosses between varieties, not species. In Mexico and in the Philippines and in Cuba there is a predominance of crosses between species of man, in the ruling classes. In this country we are now crossing species freely.

We have lost the impetus given by the original varietal hybrids. With our introduction of various conflicting elements we have not quite the freedom of which we boast, but a more or less discrete autocracy instead of such concrete autocracy as might belong to a responsible king. The politician represents as distinct a type of character as does the poet. The politician exercises a discrete autocracy in every town, hamlet and State, and we have not the freedom of which we boast.

Furthermore, we are developing that most valuable, and at the same time most dangerous, trait known as individualism. Now, when individualism develops in the expression of art, science, literature or anything in the line of human activity, it may or may not be valuable to the State, but the individualism of decline, and which has been spoken of by Colonel Arthur, is a sort of individualism which is destructive. The man believes, his wife believes, that it is well for other people to bear children. They have not

time; children do not fit well into the duties of the day, and they are inconvenient, and the consequence is, our birth-rate is falling very rapidly. In this country, with our regard for the individual rather than for the State, we are having that rapid fall in birth-rate which belongs to older countries. We are reaching our cultural limitations much sooner than we should. The birth-rate is a sort of gauge which indicates what is in the boiler of a country. We may take up the question of military training for our protection with splendid advantage in many ways. In the first place, military training allows young men to feel that by mass unit action they are working for each other and for the State. Military training gives them intelligently the idea of mass action, which will hook out the hyphen between our country and the older countries. Incidentally, it teaches them deference to authority, and self-control in the interest of personal physical health.

DISCUSSION ON THE PAPERS OF COLONEL WILLIAM H. ARTHUR AND DR. ROBERT T. MORRIS

Mr. William A. Stecher: I heartily agree with the stand taken by Colonel Arthur so far as military training for adults is concerned. But my specific trouble, as Director of Physical Education in the public schools of this city, is with the younger people, and it is that part of Colonel Arthur's paper in which he spoke of physical training beginning at fourteen years of age that I would like to modify. We have a State law in Pennsylvania which makes physical training compulsory in the schools of cities of the first and second classes. As soon, therefore, as a child enters school in Philadelphia and a few of the larger cities physical training begins. The great majority of children in our State, however, do not receive any directed physical training at all.

It appears to me that in this discussion of military training we are losing sight of a number of things. One is, that the elements of training upon which most weight is placed are not those of which most people think when speaking of military training. If you will study the training given to the youth in the European nations, you will find that in the nations standing highest in military efficiency real "military" training does not begin until the young man enters the army at nineteen or twenty years of age. But as soon as the boy—and please don't forget the girl—

enters school, premilitary training begins in the form of sufficient and effective physical training.

Physical training has not only physical, but a mental and moral side as well. It appears to me that in this recent movement for preparedness very many people are suddenly beginning to realize what we teachers have always known, *i. e.*, that physical training also has a mental and a moral end. What they are asking for under the term "military training" really should be termed "more effective physical training." We have been agitating for this for many years, and therefore when I said I agreed heartily with Colonel Arthur I meant not only that every young man should serve his country, but that his training for good citizenship should begin as soon as he enters school.

I think that specific military training in the elementary schools is entirely out of place, because I make a distinct cleavage between premilitary work and military work. You will find, if you study the question, that the European nations give much more time to physical training than we give; for instance, in the majority of American cities you will find that so-called physical training, if they have any at all, consists of allowing boys and girls to stand up between their desks and do a few arm and leg and breathing exercises. Physical training of this type amounts to very little, in fact in most cases nothing is accomplished. It also can be seen that under these conditions it is impossible to secure the results in health, vigor, smartness, etc., that are secured at West Point.

Referring to another part of the paper, I would like to say that it is not quite fair to compare the physique of the young men in our colleges with that of the men at West Point. The physical requirements needed to get into West Point are very high. I wish our colleges had the same standards. We then might have a fair comparison with the young men leaving West Point and those leaving our colleges.

I am for military training, and I believe that you as physicians can help us very much in getting the type of training fitted for the growing youth. If we had the backing of your body, and had you tell boards of education that a few minutes of physical training per day will never make a healthy boy or girl, but that every boy and girl should have from an hour to an hour and a half of physical training per day, if possible in the open air, we should at nineteen years of age have young men ready to join the army, and not such poor specimens of physical humanity described by Colonel Arthur.

Dr. A. C. Abbott: I confess to a degree of hesitation in taking part in this discussion. I permitted my name to go upon the program with some degree of reluctance because I know little or nothing about the subject, though I have certain impressions.

I cannot conceive of anyone reading that most interesting book, *The First Hundred Thousand*, without being deeply impressed. It is the story of a Scottish regiment recruited from men in all walks of life, very largely from what we would call the lower walks of life. In the beginning they consisted of an ignorant, heterogeneous mob. After a few months of training they had become a coördinated, well-disciplined, self-reliant fighting machine which, as those who read the book know, gave a good account of itself when the time came to do so.

Both Colonel Arthur and Dr. Morris have referred to an advantage which is to my mind equally important. It is that of the team work seen in a trained company of men; the acquisition of ability to take and act upon orders without losing their self-respect, and thereby the learning of how to give orders when their turn comes to give them, and as we all know has been frequent during the present European war.

To those who have not read another book, namely, Mr. Maxim's interesting *America Unprepared*, I would strongly recommend that they do so. They will equally realize that modern warfare is a matter largely of machinery. The engines of war in use today require a familiarity with their construction and their operations, and one who possesses that familiarity has, in my judgment, received what might fairly be called a liberal education. In fact, modern warfare has become a new science demanding special training.

In the course of his writing, Mr. Maxim quotes the remarks of one of our most distinguished pacifists, who suggests that in the case of threatened invasion we should open our gates and invite the invader to enter. After he had done so we should call out 100,000 of our militia, surround the invader, and annihilate him.

Could anything be more absurd! I personally have not a shadow of doubt that 10,000 veterans from the present war equipped with modern machinery would have no difficulty whatever in annihilating 100,000 of our militia in a very short time, and that, too, without much trouble to themselves.

I do not express this belief because I think that as men, and men of courage, our militia are inferior to the men of other countries, but I am convinced from all that I can read on the subject, that most of the militia is untrained and inexperienced, and except in a few instances lacks familiarity with modern machinery of warfare and would therefore be terribly handicapped in such a struggle as I have mentioned.

I am an advocate of preparedness. I believe in compulsory military service, and I believe that the real preparedness should be as full an education in the construction and workings of the modern machinery of warfare as it is possible to give.

Dr. James M. Anders: The subject discussed by Colonel Arthur and Dr. Morris should certainly be of special interest to the medical profession. I think that we as physicians should take advantage of every opportunity that presents itself to point out the health value, if nothing more, of training in our camps. In this connection I quite agree with everything that Professor Stecher has said with regard to physical training in our public schools. It has always seemed to me that this training should be carried at least to the level of competent and satisfactory industrial work, and there is no doubt in my mind that if this were done we should have better material from which to make soldiers when the young men came into military training, other things being equal. I quite agree, too, with what ex-President Roosevelt recently said, namely, that military preparedness implies industrial and economic preparedness. It is the training that boys and girls get in school that makes essentially for industrial efficiency. While testifying before the Senate Committee Col. E. F. Glenn said that military drill should begin in our public schools at the age of twelve, and should be given for twenty minutes daily for every school day of every year up to the age of sixteen years. He would not put a gun into their hands during this period and would not have them called until they reached the age of eighteen. This, it seems to me, would be a feasible plan, although not in accord with the views expressed by Prof. Stecher. I would emphasize the point that efforts at preparedness should commence as early as possible in life, whether considered from the health, industrial, economic or military stand-point.

Dr. James Tyson: There is a line of training which seems to be too elementary for consideration, but which is, nevertheless, important. How many young girls and young men know how to walk or to carry out gracefully the ordinary physical acts of life such as walking, sitting, and standing? These acts are left to chance and their subjects grow up under conditions which must favor deterioration of the anatomy and physiology of the body.

Colonel Arthur (closing): I think Dr. Morris struck the keynote of this situation when he emphasized the individualistic tendency of our people—our failure to coöperate in masses. I think Professor Stecher misunderstood me; I did not mean that at fourteen a boy should receive true military training, but such training as he would receive among the Boy Scouts and in outdoor sport and exercise.

Dr. Morris (closing): I have nothing to add, except that military training allows young people to begin to think in terms of the State instead of in terms of the individual.

THE PATHOLOGICAL AND CLINICAL ASPECTS OF THROMBO-ANGIITIS OBLITERANS[1]

By LEO BUERGER, M.D.
NEW YORK

It is not my purpose to burden you with an extensive and detailed account of the pathological and clinical aspects of the disease thrombo-angiitis obliterans, because the clinical picture and the main facts regarding the pathological lesions are certainly sufficiently known to you. I shall attempt, rather, to present the subject matter from the view-point of one who has been fortunate enough to have had a large amount of clinical material at his disposal (more than 300 cases), from which all phases of the pathology and clinical course could be investigated.

Perhaps the best general view of the morbid process, the most thorough understanding and most satisfactory conception can be had by a general comparative consideration of the pathological lesions and their corresponding clinical manifestations.

If I were asked to formulate a general concept of thrombo-angiitis obliterans, I should answer as follows: Imagine a patient seeking relief for acutely swollen superficial veins of the lower or upper extremities, of sudden advent, and with all the manifestations of an acute thrombophlebitis. Imagine this process involving a considerable portion of the distal territory of the internal saphenous vein, followed by abatement of symptoms, and consequent resolution or healing. You will be in no doubt as to the general pathology nor as to the clinical course of the condition,

[1] Read October 4, 1916.

though your estimation of the etiology will in most instances, at least, be obscure.

Transfer this picture to the deeper vascular system, over the distribution of the external and internal plantar arteries and veins, the dorsalis pedis, anterior tibial, posterior tibial, and the peroneal arteries and veins, that is, with lesions in territories where objective manifestations are absent—and you will be depicting to yourselves what corresponds to my own conception of the pathological process in the disease, thrombo-angiitis obliterans. So here, too, we postulate an acute inflammatory and thrombotic lesion, but one involving deep arteries or veins, or both, as the initial stage of the pathological anatomy.

Whereas the patient afflicted with an inflammatory and thrombotic lesion of the superficial veins presents objective signs easy of recognition, the patient suffering from thrombo-angiitis obliterans in its earlier stages may offer no objective evidences suggestive of the true nature, or of the site of the lesion. It was but in a very few cases of my own series (more than 300), that I felt justified in ascribing certain symptoms to the incipient stage of the disease. Severe, non-localizable shooting pains in the calf or foot, attended with difficulty in walking, or, possibly with tender calf muscles, with or without vasomotor symptoms and coldness in the foot, with or without obliteration of the dorsalis pedis and posterior tibial pulses, may be the only symptoms. It is only when we compare the history with the future clinical course and pathology that we can relegate such indefinite signs to the onset of the affection. In most instances, however, the patient will not seek advice for such initial symptoms, either because they are not sufficiently severe to require the attention of a physician, or, because they are incorrectly regarded as rheumatic in origin, possibly due to trauma, cold, the presence of flat or weak foot, or because they are explained on the basis of some other minor ailment.

Strange to say, patients afflicted with thrombo-angiitis obliterans may present symptoms which differ in no way from those attending the thrombophlebitis of the superficial veins, or so-called migrat-

ing phlebitis. These are the cases of thrombo-angiitis obliterans in which an acute inflammatory thrombosis involves smaller or larger portions of the external or internal saphenous vein, radial, ulnar, median cephalic or median basilic vein. Such cases are the most instructive of all, for they are the ones which afford us material for pathological study. Here the veins are accessible; portions can be surgically removed, when the lesions are in the acute inflammatory stage, and submitted to histological examination.

While the former type of case is difficult to diagnose, the variety with concomitant migrating phlebitis can be recognized by a study of the vein lesions under the microscope. If the tissue be examined when the lesions are still in the early inflammatory stage, before organization or healing has taken place, certain characteristic and specific lesions can be identified, changes which I have elsewhere described as pathognomonic for thrombo-angiitis obliterans.

Having learned that the incipient lesion of thrombo-angiitis obliterans is an acute inflammatory one, involving the arterial and venous walls, we will expect an occlusive thrombosis as the immediate sequence, and will not be surprised to learn that this stage gradually gives way to one of organization and canalization, resulting in a healed product in which the vessel becomes converted into a cord, more or less adherent to its surroundings, in which even the neighboring nerves may become agglutinated and enveloped in fibrotic vascular cord.

It is the interference with the circulatory conditions of the limbs brought about by the extensive occlusive process, that is responsible for most of the clinical manifestations of thrombo-angiitis obliterans. So that it may be safely said that patients afflicted with thrombo-angiitis obliterans do not suffer directly from the disease itself, but from the disastrous occlusive thrombosis, which signalizes Nature's method of healing a vascular lesion, that has long since disappeared.

From a study of the pathological material, and from a comparison of the lesion with the clinical history, we must conclude

that insidious or clinically unrecognizable exacerbations of the lesion may occur from time to time, so that the involvement of the vascular territory with the obliterative lesion, is a progressive one, until the summit of the organized clot reaches the popliteal; in rare cases the femoral or even the iliac. It will not occasion astonishment, therefore, that the clinical manifestations, too, become more and more serious as time goes on.

Nor must we be surprised if thrombo-angiitis obliterans simulates clinical complexes brought about by arterial occlusion from other causes. Differentiation from arteriosclerotic gangrene, intermittent claudication due to arteriosclerosis, endarteritic occlusion, and other thrombotic conditions, may at times be difficult. It is the fact that thrombo-angiitis obliterans occurs in *very young individuals*, in whom both the *vis a tergo* and the cardiac power are adequate for compensation, and in whom the vascular adaptability is elastic in its scope—it is this fact that accounts for the seemingly almost inexplicable circumstance, that gangrene occurs *so* late, or may be absent, in spite of vast and extensive obliteration of arteries and veins. It is to the development of the collateral circulation, therefore, that we owe, in part at least, the production of a very peculiar, striking, and characteristic clinical picture, recognizable even though manifestations of the acute stage of the disease, or manifestations, such as migrating phlebitis, are absent.

Clinical Symptoms. I will not go into detail concerning the clinical symptoms, for they are sufficiently well known. It may be interesting merely to make brief mention of my own routine method of physical examination, one that has stood me in good stead, in the recognition and also in the differentiation of this disease from those other that closely simulate it, and then to illustrate by lantern slides the chief feature of the pathology of the disease.[1]

My own scheme includes the investigation of the following points: (1) the general appearance of the limb in the horizontal

[1] The lantern slides are not reproduced in this paper; some have been published in previous papers on thrombo-angiitis obliterans.

position; (2) in the dependent position; (3) the presence or absence of ischemia in the elevated position; (4) the estimation of the *angle of circulatory sufficiency;* (5) pulsation in the palpable vessels, iliac, femoral, popliteal, posterior tibial, anterior tibial and dorsalis pedis in the case of the lower extremities, radial, ulnar, brachial, and axillary in the upper extremities; (6) the occurrence of *induced, reactionary rubor or erythromelia.*

1. The General Appearance of the Limb. Any departure from the normal should be noticed. The presence of fissures, ulcers, perforating ulcers, bullæ, ecchymoses, impaired nail growth, gangrenous areas, signs of infection or lymphangitis or venous thrombosis, evidences of malnutrition, such as atrophy, exceptional prominence of the bony landmarks and extensor tendons, conservation or effacement of the normal irregularities of contour through edema or through thickening of the skin and subcutaneous tissues, are features of importance. Variations from the normal color—particularly marked pallor in the horizontal position, a play of color over the foot, even in the horizontal position; cyanosis, increased redness—all these are manifestations of either impaired circulation or vasomotor disturbance.

2. With the foot in the pendent position and in the absence of inflammation, a red flush involving the toes and dorsum, as well as sole of the foot, extending upward for a variable distance, rarely farther than the ankle, is a phenomenon that is characteristic of many cases and many types of reduced circulation due to vascular obturation. This is a condition of *rubor* or *erythromelia* (Gr. *erythros* = red, *melia* = limb). It is brought about by a compensatory dilatation of the superficial capillaries, and is most characteristic of the disease, thrombo-angiitis obliterans, although also found in other arterial affections attended with closure of larger vessels. It is frequently present in arteriosclerotic and diabetic cases as well. It seems to be an effort on the part of Nature to make up for the impairment of circulation by virtue of dilatation and engorgement of the superficial capillaries. Although more striking in the pendent position, the rubor may also

be present in the horizontal position, and when continuously in evidence, may be termed *chronic rubor, chronic erythromelia*, in contra-distinction to the *reactionary rubor* that may be induced by depressing the limb after previous elevation.

3. Ischemia or Blanching. This usually sets in rapidly when the affected limb is elevated, whenever mechanical interference with the circulation is present. The extent of blanching and the rapidity with which it appears, are both valuable aids in the estimation of the amount of obstructive arterial disease. When the affected limb is cold, the tips of the toes may remain slightly blue or cyanotic. Should the blanching be slow in appearing, or very hard to determine, pressure upon the tips of the toes after the limb has been elevated for some time will demonstrate whether the part has become depleted of blood or not ("expression test"). Compression of the toes of the elevated foot in normal cases will reveal the presence of sufficient bright arterial blood (rarely slightly cyanotic), while a varying degree of ischemia, with or without marked cyanosis, will accompany obliterated or obstructed arteries.

4. The Angle of Circulatory Sufficiency. The estimation of this angle is based on the supposition that the normal limb, when elevated so as to be perpendicular to the horizontal plane, that is 180 degrees, still retains most of its color. When the circulatory mechanism is defective, and the limb is elevated to the vertical, a variable degree of blanching of the foot occurs. If the leg is then gradually depressed, *the angle at which a reddish hue returns* (angle of circulatory sufficiency) will be found to vary considerably. In some cases it will be necessary to depress the limb to the horizontal before evidences of return circulation are manifest. The angle of circulatory sufficiency would then be 90 degrees. In many cases of arterial disease, the estimation of this angle is a valuable adjuvant, not only in the recognition of the extent of the circulatory disturbance, but also in prognosis.

5. Absence of Pulsation as an Indication of Arterial Occlusion. We should be able to feel the femoral, posterior tibial, popliteal and dorsalis pedis arteries, pulsating in almost

all individuals who possess patent arteries. In rare cases the dorsalis pedis may be aberrant in its course, and therefore not palpable, or neither the dorsalis pedis nor popliteal may be accessible to the touch because of the stoutness of the patient.

To palpate the popliteal satisfactorily the patient is placed on his abdomen, lying prone. The leg is held at a right angle, that is, vertical, the patient being asked to relax the hamstring muscles. The artery is then sought in the upper half of the popliteal space, just outside of the semimembranosus and semitendinosis tendons, the fingers being pressed downward against the femur. In the upper extremities, the radial, ulnar, and brachial axillary arteries should be examined for pulsation.

The absence of pulsation is, as a rule, an indication of occlusion at the point palpated, although in rare instances, postmortem dissections have shown that the site of obliteration is somewhat higher up.

6. Reactionary Hyperemia, Rubor, or Reactionary Erythromelia. By this term we mean an *induced rubor* that manifests itself in the pendent position of the foot, after the limb has been previously elevated to the vertical. It is a physiological phenomenon that ischemia or blanching of a limb, artificially produced by an Esmarch or Martin bandage, will be followed by sudden dilatation of the capillaries of the peripheral parts, when the circulation is allowed to return. So, also, blanching will occur in a leg whose larger arteries are occluded, on mere elevation 60° to 80° above the horizontal, without the use of any artificial means. When such a blanched limb is then depressed to the pendent position, a similar induced or reactionary rubor will become manifest. This well-known manifestation may be invoked in the examination of cases in which impaired circulation due to arterial occlusion is suspected. It will be found particularly useful in cases of thrombo-angiitis obliterans, although also demonstrable in other cases of organic vascular disease. In early cases, it is especially valuable, for it may be present long before the chronic condition of *rubor or erythromelia* develops.

With this introduction I may be permitted to give you a brief

survey of the pathology of the disease, pointing out the histological lesions characteristic of the various stages of thrombo-angiitis obliterans, and also calling attention to the facts that point to the inflammatory nature of the disease, and to those observations that suggest that we are dealing with a process of microbial etiology.

In 1908 I pointed out that the name endarteritis obliterans as applied to thrombo-angiitis obliterans should be discarded, since the occlusive lesion is a thrombotic one, affecting arteries as well as veins of the extremities, and that it is independent of atherosclerosis or arteriosclerosis.

My investigations which included a thorough pathological and histological study of the vessels in 45 amputated lower extremities, 1 upper extremity, and 25 pieces of superficial veins resected and exsected from the lower and upper extremities during attacks of so-called migrating phlebitis, have demonstrated that when the patient comes to the physician for observation the larger arteries, and often the larger veins, are completely obliterated. As a rule the plantar vessels, dorsalis pedis and many of its branches, anterior tibial, posterior tibial, peroneal and sometimes of popliteal are already completely closed, although any one or more of these vessels may escape. One or both the venæ comites may partake of the same lesion. The obturating tissue is for the most part representative of, or indicative of, a healed lesion, or the end-stage of a process whose incipiency is marked by an acute inflammation of the vessel wall, with consecutive, red, occlusive thrombosis of the affected vessel. It is only in rare instances that the early stages of the vascular lesion are found in the deep vessels, but in superficial veins, when they are affected with the lesion of migrating or thrombophlebitis, the early or acute stage of the disease can be studied.

Gross Pathology. The deep vessels of the amputated legs regularly show an extensive obliteration of the larger arteries and veins. Besides this, there are two other lesions which vary greatly in their intensity, namely, the peri-arteritis and the arteriosclerosis. The appearance of the vessels on gross section depends

upon the age of the occluding process. Usually the vessel is seen to be filled with a grayish or yellowish mass that can be distinctly differentiated from the annular wall of the vessels, and that appears to be pierced at one or a number of points by an extremely fine opening through which a minute drop of blood can be squeezed. Such obturating tissue is firm in consistency, and does not at all resemble the crescentic or semilunar occluding masses typical of arteriosclerosis. The vessel itself is usually contracted, so that its wall appears somewhat thickened. This picture is characteristic of arteries or veins which are the seat of a very old obliterating process, and is to be found most frequently in the peripheral portions of the vessels, although at times this type of lesion may extend throughout the whole length of the vessel, from the dorsalis hallucis into the popliteal.

As we trace certain of the obliterated arteries or veins upward, we are apt to meet with a change in the character of the obturating tissue. Frequently it becomes softer, more brownish in color, and terminates abruptly in the lumen of an apparently normal vessel; at other times the brownish tissue gives way to soft reddish masses which are evidently the results of recent thrombosis. In some cases this thrombotic process occupies large portions of the vessel's course; in others, it is of short extent and terminates in a long cone of recent thrombus.

The veins share equally with the arteries in the lesion of occlusion. In some cases the veins are more extensively involved than the arteries, and this is particularly true of the collaterals of the posterior tibial, which are often closed when the anterior tibial veins are open. As for the arteries, we usually find an obliteration of a part or of the whole of the anterior tibial occlusion of the dorsalis pedis, and dorsalis hallucis, of the posterior tibial and plantar vessels, with or without involvement of the peroneal. Sometimes the anterior tibial is practically normal in its upper half or upper two-thirds. More rarely a large portion of the dorsalis pedis is open, with the beginning of the occlusion in the upper part of this vessel or in the lower part of the anterior tibial.

Besides the lesion of occlusion there are two other striking changes, namely, a certain amount of arteriosclerotic thickening and peri-arteritis. Arteriosclerosis is absent in the younger cases; when present, it is never pronounced, except in those rare instances in which the patient has suffered from the disease for many years, and has reached the age of forty or more. As a rule we note but a very slight degree of whitening or thickening of the intima, here and there, in the patent portions of the vessels. In a very few cases small atheromatous patches are present.

A much more interesting and more important change is the fibrotic thickening of tissues immediately about the vessels. Wherever the vessels are occluded, there is apt to be an agglutinative process which binds together the artery and its collateral veins, and sometimes also the accompanying nerve, so that liberation of the individual vessels by dissection is difficult. The adhesive condition is due to fibrous tissue growth, and varies considerably in its amount. The peri-arterial fibrosis varies, sometimes being almost absent, at other times so great, that isolation of the vessels or nerves becomes impossible, and the vascular structures make up one dense rigid cord.

Histopathology. The lesions may be considered in two stages: (1) the healed or organized stage, and (2) the acute or incipient stage of thrombosis. Between the earliest alterations in the deep arteries and veins and superficial veins and the finished product there is a large number of intermediate pictures that illustrate the metamorphosis of the obturating clot into .the intravascular cicatrix.

1. *Healed or Organized Stage.* The most common lesion is a total obliteration of the lumina of arteries and veins by connective tissue. Histologically this may be extremely varied in the general appearance, but each picture can be interpreted correctly as having its origin in the lesion of occlusive thrombosis. This obturating connective tissue usually harbors numerous small vessels, pigment containing hemosiderin, and a fair amount of connective-tissue cells. The canalizing vessels, when they become dilated ,form smaller or larger sinuses, giving the fenestrated or cribriform

lesion seen on microscopic section of the vessels, or when the canalizing vessel becomes eccentrically placed, and sufficiently large, this sinus is responsible for the appearances which have been incorrectly interpreted as the product of an endarteritis obliterans.

Elastic tissue stains demonstrate characteristic differences between this process and arteriosclerosis. Thus, the region of the organized clot is almost completely free from elastic tissue. The small amount which is present, is concentrically disposed about the new-formed vessels.

Still more suggestive and instructive is the finding of various stages of the disease in different members of the same vessel sheath. Thus, in one of the lantern slides shown, a large artery affords a view of the old lesion, as well as one of its venæ comites. Another accompanying vein, however, is in the "acute" stage of the disease, a smaller venule or satellite being in the intermediary stage, where certain "miliary giant-cell foci" make their appearance. Such pictures not only reveal the thrombotic nature of the disease, but also present an argument in favor of the following two assumptions: that the disease begins with an inflammatory lesion attended with occlusive thrombosis, and that it affects the arteries and veins in a sort of relapsing fashion, very much in the same manner as in the veins in migrating phlebitis.

The termination of the occluding tissue in arteries and veins is often seen in the form of a rounded, convex projection looking upward (cephalad), and lying in practically healthy vessel wall. At other times the old occluding tissue is capped by an additional clot which rises in pyramidal fashion, ending by a long tapering extremity.

2. *The Acute or Specific Lesion.* The early lesions are so characteristic histologically that their appearances are practically specific for thrombo-angiitis obliterans and may permit the pathologist to make a diagnosis of the disease. They are rarely to be seen in the deep vessels, for the reason that patients do not allow amputation until the disease has lasted for months or years. However, they can be well studied when these are the seat of the

typical migrating phlebitis, and have been shown by me to be identical with the acute lesions in the deep vessels.

The earliest changes appear to be the usual evidences of an acute inflammatory process involving all the coats of the vessel. The media, adventitia and perivascular tissues are infiltrated with polynuclear leukocytes and the lumen of the vessel is completely filled with red clot. In the peripheral portions of the clot, larger or smaller foci of leukocytes (purulent foci) begin to form, whose growth occurs by virtue of immigration of leukocytes. Then certain peculiar giant-cell foci develop, and are characteristic. They contain giant cells, endothelioid cells or angioblasts and numerous broken-down leukocytes. These foci then undergo connective-tissue replacement. The giant cells gradually disappear; numerous small vessels are formed, the final product being a fibrous nodule containing vessels and some pigment. In the rest of the occluding clot, the organizing process is somewhat different, resembling that which characterizes the organization of blood clot in other thromboses.

In short, the lesions in thrombo-angiitis obliterans are in chronological order: (1) an acute inflammatory lesion with occlusive thrombosis, the formation of miliary giant-cell foci, (2) the stage of organization or healing, with the disappearance of the miliary giant-cell foci, the organization and canalization of the clot, the disappearance of the inflammatory products, and (3) the development of fibrotic tissue in the adventitia that binds together the artery, vein, and nerves.

A CONSIDERATION OF THE TREATMENT OF PERIPHERAL GANGRENE, DUE TO THROMBO-ANGIITIS OBLITERANS, WITH REFERENCE TO FEMORAL VEIN LIGATION AND SODIUM CITRATE INJECTIONS[1]

By NATHANIEL GINSBURG, M.D.

The surgical treatment of impending or actual gangrene of the extremities due to pathological changes in the peripheral bloodvessels, termed thrombo-angiitis obliterans by Buerger, embraces a number of procedures which attempt to increase the circulation in the impaired limb. The successful application of a surgical principle to cure a pathological process presupposes the possibility of either totally extirpating the diseased area or structure, or the correction of a departure in normal organic function until such time as may be required for tissue restoration or regeneration to take place.

Buerger's convincing demonstration of the true pathological entity of the disease which bears his name leaves no doubt as to the presence of the extensive bloodvessel changes which have already taken place when these cases are first observed. In most instances the disease has progressed to the point of impending gangrene or actual digital death, and all surgical measures for relieving the great suffering and distress of these patients are really only palliative in nature.

A considerable amount of experimental laboratory work, performed abroad and in this country, for the purpose of establishing the value of arteriovenous anastomosis in the treatment of this

[1] Read October 4, 1916.

disease, has proved rather conclusively that true reversal of the circulation in the affected limb cannot be accomplished, even though Carrell and Guthrie successfully established in their laboratory experiments the possibility of conducting arterial blood to the peripheral capillaries by way of the veins.

Arteriovenous anastomosis, femoral vein ligation, section of the sympathetic fibers about the femoral vessels, multiple ligations of the superficial varicose veins in the affected limb (Lilienthal), and high amputation and intravenous saline injections have been proposed and performed by observers all over the world in their efforts to combat the circulatory failure in the affected limbs. In addition to these measures every conceivable form of local treatment to stay the impending or spreading gangrene has been employed with almost uniform failure in the final results obtained.

Willy Meyer has recently contributed an important article upon the employment of conservative measures in the treatment of peripheral gangrene, and refers to the interesting work of von Oppel, who has performed femoral vein ligation in these cases, with the belief that the peripheral venous stasis thereby induced from the anatomical point of view has the same relative value as arteriovenous anastomosis.

This latter operation, often incorrectly termed reversal of the circulation in the affected limb, was first performed by San Martin Y Sastrustigui in 1902, and has been exploited by Coenen, of Breslau, Wieting, of Constantinople, and many others both abroad and in this country. The operation owes its conception to the successful end-to-end suture of bloodvessels practised by Carrell and Guthrie. As originally performed it consisted in an end-to-end anastomosis in Scarpa's triangle between the femoral artery and vein attended by section of each structure and ligation of the proximal end of the vein and of the distal end of the artery. Later, lateral arteriovenous anastomosis of these vessels in the thigh was suggested by Bernheim and Stone, of Baltimore, with ligation of the femoral vein proximal to the site of the anastomosis as a necessary and important feature of the operation. As a rule the anastomosis in the thigh was made below the profunda femoris vessels.

The final results obtained, and a study of the philosophy of this surgical procedure, lead to the conclusion that the operation is founded upon a faulty anatomical and physiological basis. Guthrie has stated that he did not believe that arteriovenous anastomosis successfully applied to animals in the laboratory should be employed in the human being suffering from advanced vascular disease of the type under discussion. Stetten, of New York, in stating his views regarding the futility of arteriovenous anastomosis in the treatment of impending gangrene of the lower extremity, supplemented his condemnation of this operation by reporting convincing proof based upon injection experiments and dissections of the bloodvessels in amputated limbs. His studies have been confirmed by Horsley, of Richmond, whose conclusions are in unity with those of Coenen and Wiewiorowski, who likewise concluded, in 1911, that the operation was unsound and dangerous.

Stetten studied 136 published arteriovenous anastomoses, or attempts at this operation. He found that 30 deaths resulted immediately or shortly following the operation, and 11 patients died after amputation following the performance of the anastomosis. The immediate death-rate therefore was 30 per cent., and of those that did not die, 45 required amputation. Therefore in more than 72 per cent. of the cases arteriovenous anastomosis had either failed or could not be accomplished, owing to the technical difficulties encountered. Insufficient lumen of the vessels or advanced arteriosclerotic changes precluded even a successful attempt at bloodvessel suture in many of the failures reported.

If any value attaches to arteriovenous anastomosis, it is probable that the good resulting from the operation is solely due to the ligation of the femoral vein on the proximal side of the newly created opening, thereby practically accomplishing in principle a pure femoral-vein ligation. Meyer has recently reported a case of Buerger's disease with involvement of the base of the big toe and adjacent metatarsal bone, in which he performed arteriovenous anastomosis, with a good result. An analysis of the history of his case compares so closely with one of my own, in whom only ligation of the femoral vein was performed, as to make the cases

identical from the stand-point of involvement and successful end-result achieved. In this instance the success attending the operation is credited to the so-called "reversal of the circulation" which is believed to have resulted.

No doubt there have been many failures following arteriovenous anastomosis for peripheral gangrene which have not been reported, and I can personally add to the long list of failures one of my own, of which no previous mention has been made.

It is apparent that arteriovenous anastomosis has no place in the treatment of this disease, since the high percentage of failures will always exist, owing to the technical difficulties attending the operation and the advanced state of the disease when the operation is performed. It must be borne in mind that while these patients suffer principally from peripheral vascular disturbances, they are also poorly nourished and bear other evidence of a clinical syndrome of which their peripheral trophic disease is the most striking feature.

Femoral-vein ligation is a simple technical procedure, and will certainly and effectively produce circulatory stasis, and thereby bathe the affected limb in more blood than is present when no retardation of the venous system exists. Since Buerger has shown that the principal changes are largely confined to the peripheral arteries the tissue asphyxia resulting from deficient oxygenation is not overcome by this operation. This operation has been performed by von Oppel, Coenen, Lilienthal, Horsley and others, and by myself in 4 cases. If desirable, it may be done under local anesthesia. In effect this operation has no doubt been performed many times, since the examinations after death, following arteriovenous anastomosis, have shown thrombus occlusion at the newly created bloodvessel stoma, or contraction amounting to a complete functional closure of the opening between the artery and the vein.

Independently, or in conjunction with this operation, the employment of intravenous injections of a saline solution, either 2 per cent. sodium citrate or Ringer's solution, has been carried out, following the suggestion of Kogo and Mayesima, of Japan.

They reported 15 cases of this disease treated with injections of a saline without femoral-vein ligation in the surgical clinic of Professor Ito. Employing a viscosimeter for testing the blood, they concluded that there is an increased viscosity in the blood of patients suffering from this disease. Based upon this observation came the conclusion to employ sodium citrate solution for intravenous injection in order to decrease the degree of viscosity.

Garbat, of New York, has recently stated and proved that multiple injections of 2 per cent. sodium citrate solution into the veins of the human organism do not have a deleterious effect, and I have also employed multiple injections of this solution in varying amounts in conjunction with femoral-vein ligation.

Other than the mere mention of a single case (Lakeside Hospital in Cleveland) I have been unable to find any reference to the results obtained by section of the sympathetic fibers about the femoral vessels. Many cases of thrombo-angiitis obliterans have as a concomitant feature of the symptom-complex pronounced vasomotor disturbances, but it is difficult to conceive how the advanced thrombus occlusion of the bloodvessels can be antagonized by section of the vasomotor fibers surrounding the large bloodvessels in the thigh.

When one witnesses the excruciating pain which these patients suffer, with the attendant loss of sleep, often uncontrolled by sedatives, the temptation is quite strong to perform early amputation before or at the first evidence of peripheral digital death. Haste in amputating, without first employing conservative measures, will often sacrifice extremities which may have been saved. In many instances, even though palpable pulsation of the vessels is wanting, patients will carry limbs for years with lessened pain and arrested disease after one or more toes have been lost. If the process is rapidly gangrenous and conservative measures are contra-indicated, early high amputation should be advised to obviate the greater dangers of delay.

These cases haunt the hospitals for months and years, and one surgeon will begin by amputating a toe, and after having visited many institutions, the final termination of the disease in the

extremity or extremities involved will be high amputation by a surgeon in another institution. The first operation is not always the last, and if a follow-up system is employed in these cases, one is not surprised to learn that physical usefulness usually ceases with the early onset of the disease. One patient who came under my observation had had his first operation at the hands of Mixter, of Boston, fourteen years previously. Then followed successive operations by many surgeons in various institutions, and he finally entered the Mount Sinai Hospital with two unhealed stumps a few inches below Poupart's ligament. Employing spinal anesthesia, his long-continued suppuration was finally terminated. He had been an inmate of hospitals almost constantly since his first operation, and between his cigarette habit and the use of drugs to which he had become addicted, due to his great pain, he was in a restful state of mental apathy.

Another patient, whom I saw from the earliest physical appearance of the disease, a man, aged forty years, a carpenter by trade, suffered first the amputation of the toe of one leg, and then the leg itself was removed just below the knee, with long-continued suppuration and final slow healing of the stump, preserved for an artificial limb. A tourniquet was not required and there was scarcely any spurting from the tibial vessels when the amputation was performed. A year later he developed involvement of the big toe of the other leg, followed by slow and progressive involvement of the foot, refusing operation, until finally driven by the fearful pain in the gangrenous foot and leg to have removal of the limb in another hospital.

With reference to local treatment we have tried various saline foot-baths, heat, Bier's hyperemia, electrotherapy, and many drugs locally to the diseased parts, none of which have appeared to have any specific value. Much has been claimed for ascitic fluid applied locally to the involved digits. I personally have amputated limbs in which free use of ascitic fluid was made and have never seen any benefit result from its employment.

During the period of life in which this tragic disease is commonly observed in the male adult the female is engaged in repeated

pregnancies and thereby suffers from constant engorgement of the lower extremities as the result of increased pelvic pressure due to the gravid uterus. This might possibly be a factor of some significance in determining the discrimination of the disease in favor of the male sex. Most patients suffering from this disease are inveterate cigarette smokers, using a cigarette of cheap tobacco rolled in paper of very poor quality.

Among the causes assigned to the production of this disease are infection (Buerger), some underlying toxemia, and altered quality of the blood (Meyer). The determination of the causative factors producing a disease of this type, having sharp limitations with reference to age, habits, nativity, and sex, calls for more determined and prolonged study in order to elicit a method of treatment more successful than any now at our disposal.

With reference to femoral-vein ligation, the important facts in 4 cases are as follows:

Case I.—W. K.; aged fifty-three years; Russian; fruit dealer; admitted to the Jewish Hospital May 21, 1916. Chief complaint: excessive burning pain in the right first and second toes. In December, 1914, the patient felt a burning sensation in the right big toe associated with constant severe pain, making sleep impossible. The toe became blue and extremes of temperature applied to the foot increased the pain. Electrotherapeutic treatment at the hands of the late Dr. W. L. Rodman improved his condition, and the pain was lessened for a period of six months. In December, 1915, the pain and discoloration spread to all of his toes, involving the dorsal surface of the foot. Patient was born in Komnitz, South Russia, and came to this country when nineteen years of age. He began to smoke cigarettes when ten years of age, smoking ten to fifteen daily. He had typhoid fever at the age of sixteen; denies venereal disease; does not use alcohol; has worked hard all his life. He is married and the father of nine children, two of whom died in early infancy; the others are living and well. The winters are very severe in his birthplace, thirty miles from Odessa, and he was subjected to severe climatic exposure when he resided in Russia. Before he came to America he worked at a machine

employing both feet to run it, for about twelve years. After coming to this country he peddled fruit for two years, then worked in a fish store, finally going into the fruit business, being constantly exposed all this time to weather conditions. He has never been a big fish-eater.

Ten years ago, at the age of forty-three years, he was in the Jewish and Medico-Chirurgical Hospitals, the left foot having been amputated in the former institution for the condition which is now affecting his right lower extremity. This amputation followed multiple operations upon the foot for the relief of his condition. At the present time the pain in his right foot is persistent and unendurable without a sedative. The posterior tibial and dorsalis pedis arteries are not palpable. The mesial surface of the big toe at the first interphalangeal joint shows a trophic skin ulcer. The break in the skin is not complete. The nails of the first and second toes show marked trophic changes, the nail-bed of the second toe being the site of an ulcer of small dimensions. The dorsal veins of the foot are numerous, but are fine and not distended, and the circulatory failure in the foot is marked by a line of cyanosis about 1 cm. proximal to the metatarsophalangeal joints, and the toes are mottled and cold.

May 22. 50 c.c. of 2 per cent. sodium citrate solution injected intravenously.

May 25. 32 c.c. of 2 per cent. sodium citrate solution injected intravenously.

May 26. 75 c.c. of 2 per cent. sodium citrate solution injected intravenously into the femoral vein, with coincident ligation of this vein by silk and catgut ligatures and separate ligation of the long saphenous vein. The limb following ligation became deeply cyanotic, with intense mottling and marked diminution in temperature.

May 27. The appearance of the lower extremity is unchanged. The toes are intensely cyanotic, having a deep blue hue, and the foot is cold.

May 28. The leg is warm down to the ankle, with slight distention of the dorsal veins of the foot. The toes are less cyanotic,

the blue color having given way to a purple-red hue, sharply defined at the metatarsophalangeal junction. The patient says he has less pain and slept better last night than any night since his admission into the hospital. Over the upper third of the leg there is some venous distention, an oblique vein crossing the tibia from within and outward and downward. The dependent position of the foot hanging over the side of the bed increases the reddish-blue hue of this part.

May 29. There seems to be more prominence of the dorsal veins of the foot and the color of the toes is assuming a normal red tone. The toes, however, are still cool and the lowered temperature of the part extends to the upper third of the leg. The nail-bed of the second toe is dried up.

May 31. 100 c.c. 2 per cent. sodium citrate solution injected intravenously.

June 5. The foot is warm, there is little pain and the venous stasis has completely disappeared. The toes are still discolored and reddish blue, but there is very pronounced improvement in the circulation of the foot.

June 6. 100 c.c. of 2 per cent. sodium citrate solution injected intravenously.

June 7. Patient's leg is much warmer and he states that he feels less pain. Patient requested that further intravenous injections be discontinued and at his request was allowed to go home. (Total 357 c.c. of 2 per cent. sodium citrate injected.)

August 4. The right leg is somewhat edematous, toes are cyanotic, tense and uniformly involved in the disease. The dorsal surface of the foot is blue and very painful. There is no venous distention such as he had shortly after his operation, and apparently little permanent good has resulted from the operation. His pain has returned and is becoming severe, necessitating the constant employment of narcotics.

This case has been reported somewhat in detail because it is a typical example of a case of thrombo-angiitis obliterans relative to nativity, history and onset of the disease terminating in the loss of one extremity and the future loss of the other, following a

total occlusion of the femoral and saphenous veins with coincident injection of sodium citrate solution.

Case II.—M. G.; male; aged twenty-seven years; operator by trade; born in Russia. Admitted to Mount Sinai Hospital May 8, 1916. Diagnosis: Thrombo-angiitis obliterans of the left foot, with the presence of digital ulceration. He was treated for many weeks in the Polyclinic and Jefferson Hospitals, without relief. The details of the disease are too time-consuming to enumerate.

May 11. Ligation of the femoral vein was performed below the point of entrance of the long saphenous vein. Marked venous stasis promptly resulted, assuming the startling appearance of the case previously reported. He received daily injections of Ringer's and sodium citrate solution in large quantities from May 12 to May 19 inclusive, when he refused further injections and left the hospital against advice. The social service worker following this case traced him to another hospital and reported that amputation of the leg was necessary, owing to the progression of the disease.

Case III.—J. E.; male; aged thirty-six years; Russian; baker by trade. Admitted in Mount Sinai Hospital January 10, 1916; discharged February 8, 1916. Diagnosis: Thrombo-angiitis obliterans of the left foot. Patient came in the hospital with severe pain in the big toe of the left foot, which became gangrenous, the gangrene extending to the tarsometatarsal junction corresponding to this digit. The acute process was of six weeks' duration, the toe becoming gangrenous two weeks previous to admission. Patient was an inveterate cigarette smoker.

January 10. Disarticulation of the big toe and the metatarsal bone was done; the wound was not sutured and no ligatures were required.

January 14. The wound was suppurating and gangrene was apparently slowly spreading. Temperature, 100° to 102°. Patient discharged February 8, with a suppurating wound and marked failure in the circulation of the foot. He was readmitted to the hospital May 31 with an unhealed wound and discoloration of the remaining toes of the left foot. The toes and dorsal surface of the right foot likewise show circulatory failure. The dorsalis

pedis artery is not palpable in either foot and the veins are small and not distended.

June 3. Femoral vein ligated, with coincident injection of 100 c.c. of 2 per cent. sodium citrate solution.

June 4. Left foot is much warmer than the right; the veins are congested and the patient states that he feels more comfortable. 100 c.c. of 2 per cent. sodium citrate solution injected intravenously.

June 5. Patient is comfortable.

June 6. Patient's left foot and leg are slightly swollen, due to the venous congestion produced by the operation. Extremities quite warm to the toe-tips with normal color and very marked distention of the surface veins of the foot. The opposite foot (right), which has not been operated on thus far, is distinctly colder than the left, and is now giving much pain and discomfort, with marked cyanosis of all the toes, the big toe being especially involved. In comparison the left foot seems considerably better nourished, and the result of the ligation at this time is unquestionably astonishingly good in this case. The transformation seems almost unbelievable, and the patient is considering the same procedure in the right lower limb. He has slept better and is having practically no pain in his left foot.

August 1. Patient has been at work all summer, having little pain in the left lower extremity, which is markedly swollen, the edema having increased the leg to almost twice its normal size. The nutrition of the limb at the present time is good and the progress of the disease seems arrested. The wound resulting from the removal of the big toe and the contiguous metatarsal bone has completely healed. He is now suffering from involvement of the other extremity, and the problem of treatment arises again in this case.

The patient was observed on October 3, and the examination of his left lower extremity showed considerable diminution in the size of the limb as the result of stasis resulting from ligation of the femoral vein. He is suffering practically no pain in this limb, and complains of a pain and feeling of fatigue affecting the right lower extremity.

Case IV.—M. T.; aged fifty-eight years; was a truck driver in his native land, becoming a huckster upon his arrival in this country. Began smoking cigarettes at twelve years of age, averaging ten to twenty daily, and has been a heavy whisky drinker for many years. Married, had twelve children, eight of whom are living and well. Was in the Russian army for six years. Father died at ninety, mother at seventy-five. Family history negative in relation to the present disease. Ten years ago, when forty-eight, patient suffered from pain in the calves of both legs, compelling him to stop work at intervals. This pain continued until nine months ago, when his condition became so aggravated that he had to cease working. He has had a reddish-blue discoloration of the toes of both feet for the last six years and ulcerated areas over the anterior surface of both legs for the past five years, with failure of healing accompanied by great pain. The ulcers are trophic and pulsations in the vessels of the foot are absent. He was treated in the Polyclinic Hospital for six weeks by Dr. George P. Müller, who believed that the case was one of thrombo-angiitis obliterans, and submitted the patient to saline injections. This case was one in which there was circulatory failure, but owing to the age of the patient and the atypical location of his trophic ulcers I do not know whether he can be termed a typical case of thrombo-angiitis obliterans, although the examination of both feet confirmed the proper classification of the disease as one of this type.

May 31. 50 c.c. of 2 per cent. sodium citrate injected intravenously.

June 3. 100 c.c. of 2 per cent. sodium citrate injected intravenously into the femoral vein, accompanied by ligation of this vessel. The leg immediately assumed an intensely purple hue, with a most pronounced venous stasis of the limb below the knee-joint. The patient was in a state of shock at the conclusion of the operation, with subnormal temperature and rapid pulse, and cried out because of intense pain in the limb.

June 4. Suppression of urine was marked, only one-half ounce having been recorded in the past twenty-four hours. The right

lower limb is now mottled and markedly cyanotic below the knee-joint. The left leg still remains cold and blue. Anuria is complete, the patient having the odor of urine on his breath, is vomiting, and has a subnormal temperature.

June 5. Patient's condition remains the same. There is very marked or total venous obstruction of both lower extremities, suggestive of a thrombus of the inferior vena cava. Patient died on this day, apparently of suppression of urine.

He was a poor operative risk and undoubtedly not a good case for femoral-vein ligation.

Femoral-vein ligation based upon the experience of these few cases is an operation of doubtful value, since only one of the four patients showed any improvement following its performance. This case has unquestionably improved to the point of security of the limb in which the venous current was obstructed. Involvement of the other leg is now taking place and the value attaching to the procedure may be still greater in the future, since it may preserve one of his extremities.

The operation is an hazardous one and may cost the life of the patient, as occurred in Case IV of my series. If ligation is done, the ligature should always be placed below the entrance of the long saphenous vein into the femoral vein, thereby preserving some collateral venous circulation in the affected limb. Following this operation there is developed a large posterior femoral vein, passing from the popliteal space as a tributary to the sciatic or inferior gluteal vein.

In one of my cases ligation had no effect whatever in retarding the progress of the disease, and even failed to produce venous stasis in the affected extremity. It was a convincing example of how greatly impaired was the arterial distribution to the peripheral parts in the affected limb, and proved that the problem cannot be attacked except by dealing with the arterial element in this disturbed circulation.

I believe that the poor arterial circulation present is much better than the results attained by any of the proposed surgical measures to increase the circulation in the involved extremity.

We must bear in mind that this disease is almost invariably seen in its terminal stages, and that surgical measures are simply palliative in nature and cannot achieve a positive cure. My own experience with the injection of the sodium citrate solution has not been very satisfactory, since I have noted little improvement in the patients, and have found it difficult to continue the injections over a long period of time, since so little early relief has followed them. This experience is in agreement with the later reports dealing with the value of saline injections in this type of peripheral gangrene. Much of the early improvement noted by other observers has only been temporary, and lasting results have not been attained.

Lilienthal of New York, in a personal communication, stated that he has performed femoral-vein ligation in this disease at least a dozen times. He believed improvement has occurred in all but one of his cases.

Willy Meyer has reported a single femoral-vein ligation with failure.

There have been practically no reports of femoral-vein ligation in this disease in this country, and until a statistical study is made this method of treatment must be regarded as simply on trial. Lilienthal is most optomistic and seems to have had better results than anyone else.

DISCUSSION

Dr. David Riesman: Dr. Buerger has immortalized his name as the discoverer of a new disease syndrome. He has perfected our knowledge of the pathology and symptomatology to such a degree that it is necessary to recognize the condition described by him as a distinct clinical entity. The few cases that I have seen have caused me to feel that no other affection, unless it be malignant disease of the spine, equals it in the intolerable intensity of suffering. No one has yet offered an explanation why the disease occurs with preponderating frequency in Russian, Galician, and Polish Jews. I do not believe that typhus has anything to do with its etiology. I have always felt that tobacco and exposure were the prin-

cipal factors in its causation. These two factors, at any rate, form a reasonable explanation for the prevalence of the disease in man. Dr. Buerger's hypothesis that the disease is an infectious process finds strong support in the beautiful histological sections that he has demonstrated this evening. Perhaps in the future, by the newer methods of cultivating bacteria that are difficult to grow, it may be possible to find the responsible organism. But even if the disease is infectious, the two factors I have mentioned must be recognized as predisposing causes.

Intermittent claudication occurs not only in thrombo-angiitis obliterans, but also in other conditions, especially in the common variety of arteriosclerosis. It may likewise be a purely functional condition dependent upon a temporary spasm of the arteries. Intermittent claudication may occur in women.

In the early stages of thrombo-angiitis obliterans and of intermittent claudication, erroneous diagnoses are often made. The patient is supposed to have chronic rheumatism or flat-foot. To avoid mistakes it is a good practice in all cases of pain in the feet to examine the dorsalis pedis pulse. It is so nearly constant in health that its absence should at once suggest the existence either of arterial disease such as thrombo-angiitis obliterans or less often intense vasomotor spasm.

In regard to treatment, there is one point to which I should like to call attention, namely, that surgeons should be rather slow in resorting to amputation or other radical measures, such as ligation of the femoral vein, until it has been definitely established that the obstruction is organic. In one case that I have seen in which two surgeons advised amputation, the circulation was restored in the limb and the patient regained full power of walking.

Dr. George P. Müller: I think Dr. Riesman failed to mention one disease, the name of which originated here. I refer to erythromelalgia, sometimes spoken of as Weir Mitchell's disease. Several cases of this disease were referred to Dr. Frazier's clinic years ago, and it was through observation of these that I subsequently became interested in Buerger's disease, although, of course, there is no similarity between the two, except in the terminal stages.

In the fall of 1909 I performed an arteriovenous anastomosis for threatened gangrene of the foot and reported the case (*Annals of Surgery*, 1910, li, 247). I think there were only twelve cases on record at that time. Since then many others have been reported, and in 1915 Stetton reported 136 cases. After analyzing them and excluding many of the so-called successes, he concluded that 16 of the number may be considered to have been successful. Of these 11 were reported by four men.

In 1910 I offered the following conclusions:

1. "That in the early stages of arterial disease producing ulcers on the toes, erythromelia, extreme pain, tingling, etc., a complete reversal of the circulation *may* relieve the condition if other measures have been tried and failed." I do not remember whether I actually believed at that time that a complete reversal of the circulation could be effected. At any rate I do not now believe that it can be accomplished. The result of operation probably depends upon the congestion produced by the ligature of the femoral vein. It hardly seems possible that the blood will be forced through the thin-walled veins clear to the toes. The very function of the vein is opposed to this possibility, and in Buerger's disease the veins are thrombosed. It is uncertain whether arterial blood going through the veins will give nourishment to the tissues.

2. "That with gangrene of a toe established one should wait for a line of demarcation. If the process involves several toes or tends to spread to the dorsum of the foot, an anastomosis between the femoral artery and vein with ligation of the external saphenous *will almost certainly* induce a line of demarcation in the region of the ankle." I certainly believe that it is possible for the valves in the popliteal vein to be forced and for the blood to reach a certain distance through the veins. The area of the leg is thus kept flooded with blood and the extension of the gangrene from the foot upward may be avoided. The ligation of the femoral vein again produces a congestion and an increase of blood in the tissues threatened with gangrene.

3. "That if the superficial and deep veins are also thrombosed the operation is useless and should not be done." I have not altered my first opinion, namely, that in Buerger's disease, arteriovenous anastomosis is useless.

Finally, in conclusion, I believe that the merits of the operation have been distinctly overrated and that the so-called successes are not nearly so successful as their recorders would have us believe.

Dr. George M. Dorrance: In our work upon the coagulation time of the blood we found that the citrate solution did not have any effect because of the constant tendency of the blood to return to normal. I think many of the operations for this condition have gained their reputations from the periodicity of the cases and not from the operations themselves. I have not performed any reversal of circulation upon the human. It has been definitely proved that the blood returns to the heart by the first large vein below the arteriovenous anastomosis. This being so, I cannot see the least justification for the performance. Technically it is frequently impossible to perform on account of the sclerosis of the

artery. The ligation of the veins seems to be the operation indicated at present.

Amputation of the thigh in the lower third is the operation I advise, so that the patient may return to work and be free from pain.

Dr. Buerger (closing): I think Dr. Dorrance's view-point coincides very nearly with my own. I am not at all in favor of the various palliative operative procedures, because I do not believe that we can materially influence the progress of this disease. How can we hope to permanently alter a condition which is dependent not only upon the progress of the obliterating process, but also upon circulatory demands made upon the collaterals at any given moment? We are dealing with a purely mechanical proposition, and we are uncertain as to what demands will be made upon the circulatory apparatus of the limb. How can we permanently enhance the circulation through injections of saline, whose effect is evanescent? We expect to improve the vascular paths by deflecting blood through veins that are in many places already closed. The cases which reported improvement after each measures have since come back, the trophic disturbances of the disease having since become more severe. In short, none of the palliative operative measures have in my own experience successfully influenced the condition.

There is one method of treatment from which we do get some benefit. It is based upon the fact that it is possible to obtain a good reactionary hyperemia in such limbs. If we elevate and then depress the affected part, marked hyperemia results. This phenomenon can be made use of as a method of treatment which I call the postural treatment. I tell the nurse in charge of a case to elevate the limb upon a support for two or three minutes to produce the characteristic preliminary blanching. The patient then sits on the edge of the bed and allows the leg to hang over, thus producing an intense hyperemia of the foot. The leg is then put into the horizontal position for a period of rest, varying from two to five minutes. This treatment occupies about ten minutes, three minutes up, three down, and three to four in the horizontal position. This is repeated five or six times, the total duration of each séance being about one hour. Two or three séances may be given daily. If there were some apparatus in which we could place the patient's limb, and in which the limb could be automatically raised and lowered, the treatment could be carried on for hours through many days. In this way I believe we could get some appreciable result, because we would be constantly flushing the collaterals with blood.

Regarding typhus as an etiological factor, I have inquired into the history of many cases since Goodman published his paper. In none of

the cases could I elicit a history of typhus. Baehr and Plotz in their large experience with typhus in Serbia saw no cases comparable to those which we call thrombo-angiitis obliterans.

Although we have not been able to detect any organism in the tissues obtained from the acute lesions, it is possible that something may still be found in lesions that are still younger than those that were at our disposal.

It must not be forgotten, too, that other factors may act as predisposing causes. Stasis may act in making the lower extremities the seat of predilection. Tobacco may render the vessels susceptible, thus explaining the almost exclusive involvement of males. It occurs in the Gentile as well as in the Jew. I have seen three cases in which the patients were born in America. Many of these patients of Russian parentage, have never eaten rye bread. One per cent. of cases in my series has never smoked. It is to be hoped that the causative agent—microbial or other—and all the predisposing factors may become definitely recognized after additional observations on the pathology and bacteriology of the "acute lesions" will have been made.

Dr. Ginsburg (closing): I feel that there is still something to be said in favor of the femoral-vein ligation. In the case referred to I was careful to state that operation may not have done any good, but the process might have been terminal without it. Dr. Willy Meyer stated to me that he had had one femoral-vein ligation which was a failure. Lilienthal said that he had done a dozen femoral-vein ligations, that every case had been improved except one, and in that the process had been arrested. While not able to say that femoral-vein ligation has done any good, still it deserves a trial.

INTERMEDIARY METABOLISM IN DIABETES[1]

By GRAHAM LUSK
NEW YORK

The phenomena of human life are dependent upon the reactions which take place between myriads of cells and their food and oxygen supply. One kilogram of body substance contains 30 grams of tissue nitrogen. The essential waste of this nitrogen amounts to 1.7 mgs. per kilogram of body substance per hour, or one part in 18,000 of that which is contained in the body. This is the wear and tear metabolism of protein at a minimal level. One may calculate that the least amount of energy which is necessary to maintain the life processes of 1 kg. of body substance is 1 calorie per hour, which represents the quantity of energy contained in 215 mgs. of glucose. It has been estimated that 1 kg. of body tissue contains cells having a total surface area of 150 square meters. Therefore, when each square meter of this cell surface is supplied with a sugar solution containing 1.5 mgs. of glucose per hour, the life of the tissue can be maintained. One can imagine how thin a film of glucose solution around the cell is necessary for the maintenance of its life. In ordinary daily life two-thirds of the energy which supports our bodies is derived from glucose. In diabetes the power to oxidize glucose is lost. It may be interesting to inquire into the nature of the process of the oxidation of glucose.

There are two biological reactions of glucose which have long been known under the heads of lactic acid and alcoholic fermentation. In the first instance a molecule of glucose containing six carbon atoms is broken into two molecules of lactic acid, each

[1] Read November, 1916.

containing three carbon atoms. In the second instance a molecule of glucose liberates two molecules of alcohol, each containing two carbon atoms and two molecules of carbon dioxide. Both of these transformations are anaërobic; that is to say, they do not require oxygen for their production.

Lobrey de Brun and van Eckstein found that when glucose solutions contained even traces of hydroxyl ions, this alkalinity served to transform optically active glucose into a mixture of a multitude of other sugars, a mixture which did not rotate polarized light. This phenomenon was called mutarotation. Nef stated in 1907 that any ordinary hexose (such as glucose, fructose, galactose) when treated with weak alkali can yield in theory 116 different substances. Of these he was able to identify 93, of which 47 were different forms of sugar, and the rest different fragments of sugar cleavages. L. J. Henderson finds that a glucose solution undergoes mutarotation when the alkalinity of the solution is the same as that of the blood.

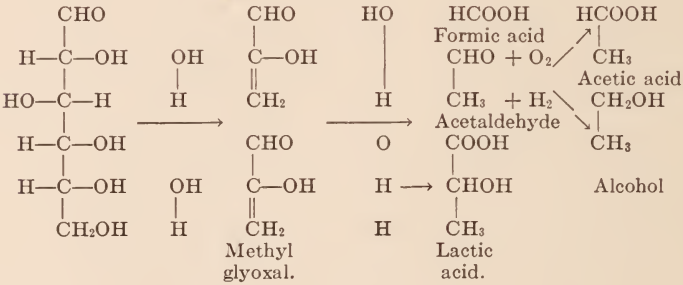
The presence of traces of acids prevents all these transformations. That this may have biological significance is suggested by the work of Rona and Wilenko, who found that a decrease in the alkalinity of a perfusing fluid greatly reduced the utilization of glucose by an excised, beating heart. They suggested that the diabetic condition is the outcome of a slightly acid reaction within the cells. The biological chemical reactions of sugar in the organism may be best explained by assuming that sugar is first dissociated into substances containing three atoms of carbon, such as methyl glyoxal or glyceric aldehyde. It is but a step to convert such fragments into either lactic acid or into alcohol; or to oxidize them; or by synthetic reunion to convert them into sugar once more. In the body the synthetic reunion always takes the form of glucose. After this fashion one can understand how levulose (fructose) may be transformed into glucose in the diabetic organism by the union of two intermediary substances, each containing three carbon atom chains.

Dakin, Neuberg and others have produced evidence which indicates that methyl glyoxal is an important intermediary product

in sugar metabolism. Methyl glyoxal is a substance which in asphyxial conditions is readily converted into lactic acid. Biologists find that lactic acid is not oxidized. On the other hand, methyl glyoxal in the presence of oxygen readily undergoes decomposition. Neuberg has shown that the yeast cell may produce acetaldehyde from it, and that this may then be reduced to alcohol. In the presence of oxygen, however, acetaldehyde may be converted into acetic acid, which can readily be oxidized in the body into carbon dioxide and water.

The provisions in this scheme of glucose metabolism are such that if asphyxial conditions are encountered, lactic acid is produced (which may be reconstructed into glucose). Under asphyxial conditions acetaldehyde does not arise in the tissues, for if it did arise in the presence of asphyxia, alcohol would then be a product of metabolism. The prior formation of lactic acid therefore automatically prevents the organism from becoming a brewery.

These relations are set forth in the following formula:



In diabetes the glucose molecule appears to be invulnerable. If lactic acid or methyl glyoxal are administered, they are completely transformed into glucose. If in diabetes the muscles be rendered asphyxial by means of strychnin convulsions, no lactic acid arises from glucose, as occurs in a normal animal, which indicates that no methyl glyoxal is formed. It is conceivable, however, that after giving fructose to a diabetic animal during strychnin convulsions, some of the dissociated three-carbon atom chains which are usually transformed into glucose might produce lactic acid.

In complete diabetes, however, fructose is limited in its break-down to chains of three carbon atoms, which are then convertible into glucose. The phenomena of the mutarotation of sugars in diabetes follows only one direction, and that leads to transformation into a single end product, glucose.

By the same method glucose is formed from protein. In the metabolism of protein, chains containing three carbon atoms are liberated in the organism, and these may be synthetized into glucose. The amino-acid alanin affords the simplest expression of this reaction. Alanin on hydrolysis is converted into lactic acid,

$$CH_3CHNH_2COOH + HOH = CH_3CHOHCOOH + NH_3$$

and, as shown above, lactic acid is convertible into glucose. A full analysis of these conditions has elsewhere been attempted.[1]

It has been established that in a fasting, meat-fed dog rendered completely diabetic by phlorhizin, there is a constant relation between the number of grams of urinary nitrogen and of urinary glucose. This relation is, Dextrose : Nitrogen = 3.65 : 1—and is commonly called the D : N ratio. Since 1 gram of urinary nitrogen represents the destruction of 6.25 grams of protein, it follows that 3.65 grams of glucose must have arisen from the metabolism of 6.25 grams of protein, or 58 per cent. of the molecular complex. It is well, perhaps, to place on record that this relation has been established for a man (a cancer patient) under the influence of phlorhizin and for severe types of diabetes mellitus. The following table verifies this statement, the D : N ratios being given in successive day-to-day periods:

D : N Ratios.

	Phlorhizin.		Diabetes mellitus in man				
Author.	In dog. Lusk.	In man. Benedict, S. R.	Mandel and Lusk.	Greenwald.	Foster.	Mosenthal.	Joslin.
	3.65	3.58	3.60	3.75	3.58	3.75	3.69
	3.66	3.82	3.65	3.56	3.38	3.85	3.67
	3.62	3.66	3.66	3.70	....	3.44	3.67
	3.64	3.68	3.64	3.64	3.48	3.66	3.68

It may be stated, without qualification, that sugar does not arise from the metabolism of fat in the organism.

[1] Lusk, Elements of the Science of Nutrition, third edition, 1917 (in press).

In complete diabetes the patient is therefore thrown upon the energy content of fat and of those fragments of protein which do not pass over into glucose for the maintenance of his life.

That this is true may be further verified by examining the respiratory quotients obtained in diabetes. It will be remembered that when glucose is oxidized in the organism, one volume of oxygen is inspired for each volume of carbon dioxide expired, and the respiratory quotient is unity. When protein is oxidized the respiratory quotient is 0.8 and fat yields a quotient of 0.707. But when glucose arising from protein cannot be oxidized the respiratory quotient of protein falls to 0.63. The respiratory quotient in severe diabetes will therefore fall below that of fat. As a matter of fact, results have been obtained which show that the quotient observed in severe diabetes is about 0.69. The calculated "non-protein respiratory quotient" is obtained by deducting the influence which the protein metabolism would have exercised upon the observed respiratory quotient. This is found to be about 0.70 instead of 0.707, the theoretical value of fat. The "non-protein respiratory quotient" would have been higher than 0.707 if glucose had been oxidized with fat. Since the analytical methods do not allow of an accuracy greater than 1 per cent., it may be deduced that the severe diabetic subsists upon fat and those fragments of amino-acids which do not form glucose.

These results appear in the following table:

	D : N.	Respiratory quotient.	Non-protein respiratory quotient.
Phlorhizinized dog	3.54	0.687	0.704
Diabetic man (G. S.)	3.50	0.697	0.700
Diabetic man (C. K.)	3.97	0.687	0.699

It may be added that the formation of β-oxybutyric acid in the oxidation of fat likewise tends to depress the respiratory quotient unless this effect is compensated for in the expulsion of carbon dioxide from its union with bicarbonate of soda.

The heat production in diabetes does not vary greatly from that found in health unless an increased protein metabolism instigates a higher oxidation. The metabolism in severe diabetes is therefore finely adjusted and follows known biological laws.

SOME CLINICAL PHASES OF DIABETES[1]

By FREDERICK M. ALLEN, M.D.
NEW YORK

I SHALL sketch some clinical experiences with diabetes. I had expected first to speak upon experimental diabetes, but changed to the clinical topic, and my remarks will be entirely informal and without any very orderly arrangement. A rather interesting case has lately come into the hospital, that of a young child in coma. The child was treated by fasting and a small dosage of alkali intravenously. At first things seemed to be at a stand-still; the carbon dioxide of the blood held about even. A little vomiting gave the hint and the stomach was washed out, showing large quantities of material, and after that the child seemed to be improved. A noteworthy feature was the lipemia present. In Dr. Joslin's book you will see in the frontispiece an illustration of diabetic lipemia, in which the blood looks like cocoa and the plasma like cream. I shall not go into the discussion of lipemia in general, but say merely that it is a condition occurring more or less in other circumstances, but never approaching in degree the true diabetic lipemia. The maximum figures on record are those of Frugoni and Marchetti, who claimed that three days before the death of a thirteen-year-old boy in coma they obtained 27 per cent. of fat in the blood and at autopsy of the patient there was 34 per cent. of fat in the blood. These figures are questioned a little in Germany, but there is no doubt of the analyses of Neisser and Derlin which showed 19.7 per cent., and of B. Fischer which showed 18.13 per cent. of fat. There are many other analyses

[1] Read November 1, 1916.

showing somewhat lower figures. In the small sample of plasma which I have here you can see the opacity and the slight yellowness. This plasma contains 11 per cent. of fat. Lipemia has previously been a sign of bad prognosis. In a number of our cases we have seen lipemia clear up on fasting, sometimes on the first fast, sometimes only later in the treatment. It is a curious feature of unknown etiology occurring in severe diabetes but not necessarily in every case; the majority of severe cases show nothing like this quantity of fat in the blood.

In connection with this I should like also to illustrate a test which Miss Wishart has introduced in regard to acidosis. It is simply the application to the plasma of the well-known Rothera test for acetone in urine, one of the modifications of the sodium nitroprusside test. The attempt seemed unpromising in view of all the substances known to be present in plasma. The plasma is first saturated with ammonium sulphate in solid form, then four or five drops of a 5 per cent. sodium nitroprusside solution is added, and finally a little ammonia. The curious thing is that the other substances in the plasma seem not to interfere, and one gets a decided acetone test in the plasma in cases of acidosis. The test gives a quick general idea of the conditions, and in cases of threatened coma a strong reaction for acetone is given in the plasma. In some cases there will be a heavy excretion of acetone and little or no reaction in the blood. In other cases it may be dammed up in the body and the qualitative test shows that it is high in the blood.

In the case of Geyelin and Du Bois, shown in Dr. Lusk's chart, the diabetes was apparently the most intense ever recorded in the literature. The man was getting no food in the earlier part of this period, and the carbon dioxide was down dangerously in spite of the large quantities of bicarbonate. He had to be fed, and as usual he improved clinically. We have seen several cases in which the initial fasting is badly borne. In every instance in which the patient has been fed a regulated diet for a number of days and then placed upon a second fast, he has always stood the second fast well. In a case such as this, a patient in the prime of

strength, a foot-ball player, suddenly developing severe diabetes, no matter how intense the rush of symptoms, a good prognosis may generally be given when proper treatment is begun early. In this case the tolerance finally rose to 200 grams of carbohydrate.

Another type of case more difficult in the long run is shown in the record of a woman, aged forty-three years, who came in with severe diabetes. She had been well until April, 1915, when she noticed excessive thirst and loss of weight. In June of the same year she consulted a physician and the diagnosis of diabetes was made. A fast of a day or two was given; then a diet of eggs and milk. On this she became not quite sugar-free. She dragged along in this condition until in August, 1915, she consulted a practitioner with considerable experience in this method of treatment, in fact, one of the most experienced, aside from Dr. Joslin. He put her upon more radical treatment, but neither the glycosuria nor the acidosis cleared up satisfactorily. She was losing weight all the time and acidosis was present, there being no carbohydrate tolerance to correct this. She went along on low diet, and was eating something like three eggs a day and several ounces of spinach, celery, lettuce, and string beans daily for six months, when the doctor, being puzzled, brought her in to us. She had never been a fleshy woman, but she came in weighing 36 kilos (about 80 pounds), having lost 60 pounds; and she had heavy acidosis and sugar excretion of about 20 grams on this low diet. A case of such severity, treated for a considerable length of time just sufficiently to keep the patient alive and yet not enough to correct the condition, is about the worst type of case for ultimate prognosis one can meet. She was placed first upon a fast of three days. The condition did not clear up satisfactorily. Then the member of the staff who was treating her thought he would like to try an experiment. This consisted in trying to build up her strength a little by means of a few square meals—a natural temptation in such cases, but the attempt always fails. She was given a diet of from 1500 to 2000 total calories a day, which is not an excessive diabetic diet. In four days her ammonia went up to about 4 grams, the carbon dioxide capacity of her plasma came

down to about 25, her blood-sugar went up along with the urinary findings. She was right on the edge of coma as the result of a very moderate diet. There was then nothing to do but to fast her. This was done, and with the help of very little bicarbonate the carbon dioxide capacity promptly climbed above the danger level. There were nine days in which she received little but alcohol in that weak condition. Her sugar cleared up; her ferric chloride reaction did not clear up. Then a carbohydrate-free diet was begun; the alcohol was continued and there was a little protein and a little fat added. Throughout all this period the total calories numbered 700 to 800 daily. This shows upon how low a diet a patient in an emaciated condition can exist. The carbon dioxide remained satisfactory and the patient's weight increased. The gain in weight was an illustration of the edema sometimes appearing in severely diabetic patients when they are cleared up in this manner. The blood-sugar remained lower than before; the ammonia was higher than it should be; there were no threatening symptoms. Here the diet, especially of fat, was slightly increased, but never above 1400 calories, and as sugar kept reappearing, was cut down so that about 1000 calories would be called the average. Traces of sugar and a positive ferric chloride reaction persisted from January to April. A low diet was continued with all the protein she could stand and as much alcohol as was advisable, and little fat, in the absence of the carbohydrate which had to be withdrawn to keep glycosuria absent. From here on the ferric chloride reaction was negative, but a trace of sugar kept cropping up. With this low caloric value we made further subtraction by a fast-day once a week. This is a routine measure in cases of that type. The first of July she was allowed to go home because she was homesick. As a result of her six months' treatment in the hospital her weight had been reduced from 36 to 33 kilos, which was about one-half of her weight in health. To repay for that her carbon dioxide was normal; her blood-sugar was down below 0.15 per cent. and her ammonia was only a little above normal. Since going home she has been able to do her housework most of the time, although showing traces

of sugar from time to time. She knows perfectly well how to take care of her own diet and has kept herself on 1000 calories a day most of this time, with a fast-day once a week, recently on only 800 calories with a fast-day every five days. Recently traces of sugar have been appearing more frequently and in a short time she will come back to the hospital for further treatment. She will come back in better condition than at her previous admission. That is the best that can be claimed for that type of case.

As to the method of making such a diet tolerable, various plans can be employed, principally in the use of thrice-boiled vegetables, which contain very little nutritive value. For bread we have the biscuits or muffins which we make out of nothing but bran, agar and salt. They contain no nutritive value nor have any taste. They can, however, be heated in the oven with butter on them. They can be eaten with a fried egg or taken with soup or food which gives the flavor. In that way the patient receives the added bulk and is much better satisfied to have his egg or bacon, or whatever else he takes, outside the muffins than inside. In this way it seems a great deal more. Food given in this way keeps the patient from being hungry to an intolerable point.

The prognosis in such a case is presumably bad. I am not convinced that diabetes is inherently progressive, that there is something inside the patient which continues sending him down. The records of our cases do not indicate that. Overstrain is a potent factor in the continuance of the disorder. Some patients improve surprisingly. I am not certain that this patient may not improve, but no claim can be made for the ultimate prognosis in such a case. I think it is probable that she will go into coma if her diet be made liberal. On the treatment outlined, however, she has been kept not only alive but reasonably contented during these six months. Before we can expect to help such a case very much there ought to be something further in the way of treatment. The only person who has a chance to do the most good for such a case is the physician who first sees the patient. If this patient had not been allowed to drag along for six months as she was, the condition would not have been so hopeless. While

at the onset by no means severe, diabetes of ordinary type allowed to drag along become severe and is very difficult to treat. I have selected such a case simply to give you a hint of what may be done under such circumstances.

DISCUSSION ON THE PAPERS OF DRS. LUSK AND ALLEN

Dr. Alonzo E. Taylor: It is impossible in a discussion of so large a subject as diabetes to do more than present briefly a few points. The clinician, even of the most advanced modern type, who views the work that for the past ten years has been devoted to the intermediary metabolism of diabetes ought not to obtain the notion that this matter comprehends the substance of the disease entirely, and that upon the elucidation of the intermediary metabolism now under investigation depends our knowledge of the pathogenesis of the disease. Certainly, the laboratory investigator has no such conception. If up to the present the laboratory investigations have laid special stress upon the intermediary metabolism, it is because it is the most suitable phase for investigation. Nearly all the studies deal with abnormalities in the catabolism of fat and sugar because these reactions lend themselves to investigation. But there is a broader view-point that every laboratory man must recognize, and which every clinician should understand, which may explain many of the divergent features of diabetes. The up-building processes of the body can never be dissociated from the pulling-down processes. There is no such thing as a disturbance in the burning of sugar without an effect upon the anabolism of sugar in the tissues, and likewise no disturbance in the burning of fat without similar influence in the building-up process of fat. Fat and sugar are vital in the building up of metabolism. We have every reason to believe that when the body cannot burn sugar and fat it cannot utilize sugar and fat in constructive anabolism. Abnormalities in the utilization of sugar and fat in the building-up processes may be as important in the production of certain symptoms of diabetes dealing with resistance as are the abnormalities in the catabolism.

I would, in the second place, draw attention to one point in connection with the current use of the Allen treatment which is based upon a misconception. Dr. Allen has not committed this misconception, but it has been done by so competent a man as Joslin. When the diabetic has been made sugar- and acid-free, how far shall he continue his diet? Shall his

increase of food be controlled by the urinary signs or shall he adopt other criteria? It has not been demonstrated that it is necessary to give the usually stated 40 calories per kilo. Investigations have recently shown that a man of 70 kilos may live sixty days upon a diet of coarse bread, potatoes, cheese, and eggs, containing about 2000 calories, without loss of weight. If such a man should happen to have diabetes and were subjected to the Allen treatment, it would be an absurdity to attempt to feed him back to 40 calories per kilo. The man dealing with a patient should bear in mind that what he needs to feed to is not the normally high maximum of calories but the low minimum standard of calories.

Dr. John H. Jopson: I wish to call your attention to some practical phases of the subject of diabetes which present themselves to the surgeon called upon to deal with surgical lesions associated with this disease. Our views as to the prognosis and treatment of the rather numerous lesions of this type have undergone some violent changes in the short time which has elapsed since the introduction of the new era of treatment of this grave disorder. We are still in need of additional light on many subjects to avoid the pitfalls which are not infrequently encountered in the treatment of such cases. In no other class of cases is the constant coöperation of surgeon and internist more necessary or more intimate. The problems which present themselves are often complex and difficult and require a knowledge of dietetics and laboratory technic which is usually beyond the limitations of the surgeon himself. Certain plain principles have been evolved which are not difficult of comprehension, and certain mistakes seem to be common enough for the results of which the surgeon is often considered responsible. It is desirable to make some classification of the surgical lesions encountered in diabetics. This has been done recently by Strouse and years ago by Phillips. Strouse divides them into emergency cases and those not of an urgent nature, and again into those in which the surgical indications are directly the result of diabetes and those in which the association is an independent one. The latter class he again divides into those with and those without the complication of infection. Phillips classifies surgical lesions as follows:

1. Cases of diabetes caused by surgical affections.
2. Cases in which diabetes causes the surgical lesion.
3. Cases in which diabetes and surgical conditions occur independently in the same individual.
4. Cases in which diabetes adds to the danger of a surgical condition.

Examples of these different groups can be readily brought to mind. In all cases in which the element of infection is present, whether accidentally associated with diabetes or predisposed to by this disease, the association

lends the gravest aspect to what may seem at first to be a trifling lesion and the two conditions act and react upon each other in a way which renders prognosis doubtful or gloomy and treatment difficult and fraught with added anxiety and danger. What are the problems associated with operation itself? It cannot be denied that the diabetic presents a poor surgical risk in these days when we are reducing our operative mortality to a minimum. The mortality ranges in different series of cases from 7 per cent. to 30 per cent. It would appear that in all cases in which close coöperation of physician and surgeon is obtained, and especially when time is given for preparation of the patient for the hazard of operation, that the results are greatly improved, the mortality being decreased by half or even more over the old days when treatment was administered in a haphazard manner or ignored altogether. For this improvement much thanks is due to our guests of the evening, Doctors Allen and Lusk. What are the dangers of operation? The first and most important are acidosis and coma. Many patients present themselves with evidences of acidosis due to the diabetic condition alone when untreated or superinduced and aggravated by the surgical condition, especially when associated with a septic lesion. It is often brought on or aggravated by incorrect methods of preparation for operation, as by sudden change in diet, especially in sick and elderly persons, by the sudden withdrawal of carbohydrates from the diet, and by injudicious starvation in an attempt to render the urine sugar-free. This antedates operation itself. The use of ether and chloroform as general anesthetics or the old practice of using strong antiseptic solutions are attended by increased mortality or morbidity, and beyond and above these the distress and shock of any major operation, even in what are regarded as mild cases, as Addis says "is something incalculable and mysterious which upsets prognosis, however carefully the evidence is balanced."

In the after-treatment poor results are often due to a neglect of the nutrition of the patient, and a hard-and-fast adherence to rules of practice which may bring about exhaustion, causing him to sink slowly but surely, and showing itself locally in failure of wound healing and progress of infection, and constitutionally by increasing asthenia and perhaps after all death in coma.

The plan of preparation which seems to be attended by the best results in these cases, when time for such preparation is permitted, is by the gradual withdrawal first of fats and proteins and then of the carbohydrates from the diet, avoiding in this way the development of acidosis. Then a period of fasting followed by the gradual return before operation to a mixed diet, first adding carbohydrates and then proteins and fat, avoiding, however, sudden changes in diet as far as possible. When opera-

tion is urgently called for in certain emergencies, perhaps in the presence of acidosis, the problem is very grave and the danger very great. We are directed to administer carbohydrates to overcome the acidosis in these cases. Our experience has been that sudden restrictions of diet and attempts at the elimination of sugar from the urine in these cases have not yielded as good results either before or after operation as has the plan of feeding the patient, and we have been especially struck with the failure of nutrition by too early attempts to control the glycosuria after operation and by the development of acidosis as a result of such attempts. We strongly urge the use of nitrous oxide and oxygen when general anesthesia is necessary, believing the use of ether or chloroform is unjustifiable in cases with glycosuria or presenting a history of glycosuria. Conservation in operation as emphasized by Stettin is often a safe plan. In four out of the six cases of amputation of the lower extremity for diabetic gangrene we did a conservative amputation in the middle of the leg rather than amputation above the knee. Three of these cases recovered, the fourth dying of gas-bacillus infection which had been present before operation. It is especially in regard to the handling of these acute cases and the time at which it is safe to treat the diabetes afterward that we would direct our inquiries at this favorable opportunity.

Dr. James Tyson: There are one or two questions which, though of an elementary character, I would like to ask of the speakers because of their eminence and the work they have done on this subject.

The first of these is, "Does the previous duration of the disease have any influence on the effect of the starvation treatment?" Is a case of long duration—say of ten years, such as I now have under observation, more likely to be cured by the starvation treatment than one which has lasted for only one year?

The second question is, "In the event the sugar can be gotten out by the old-fashioned strict diet, and can be kept out of the urine, are the possibilities of recovery any less than when the sugar is removed by the starvation method?"

Dr. Lusk (closing): With regard to the lowered metabolism in the emaciated, cured diabetic, take the case of Geyelin and DuBois. When the patient was intensely diabetic, the number of calories produced per hour, as measured by the calorimeter, was 73.2. The weight of the patient was 56½ kilograms. The heat production was normal for that weight. Later, through the starvation, the weight fell from 56½ kilograms to 46 kilograms and the man developed a high degree of tolerance for carbohydrate. The calories produced per hour fell from 76.4 to 43, or was 35 per cent.

under the normal for the lower body weight. He had fallen to a level of metabolism which was under the normal because of excessive emaciation. Thus he required only about 60 per cent. of the food that he had required previously when he was heavier and diabetic.

Dr. Allen (closing): I should like first to express my very hearty agreement with Dr. Taylor's remarks.

In regard to the questions bearing upon surgical cases—I know very little about surgery. We have not a surgical hospital and I have seen very few surgical complications. There are doubtless a variety of measures that may be required to suit various conditions. Probably the best preparation for operation is with carbohydrate, somewhat like a modified oat cure, but without the injurious overtaxing of the tolerance by excessive quantities. The patient might be made sugar-free and then for a period be given no food but carbohydrate, increasing the amount day by day until there is a slight glycosuria. This probably offers the greatest safety in operation, although no operation is fully safe. We have sometimes learned of patients after leaving the hospital going under anesthesia and through operations safely. One patient died suddenly after an appendectomy. We were not consulted and do not know the exact circumstances. This patient had severe diabetes yet was in good condition, with urine normal. A surgeon might suppose the diabetes to be imaginary or trivial. I know of one poor woman who went to a doctor for a bad cold. She told him she had diabetes. The doctor declared she had not; she insisted she had. She had to pay his bill but would not take his medicine. With such cases a careless surgeon is liable to get into trouble, since an anesthetic may send them directly into coma.

A bad diabetes and a bad infection will often make a fatal combination, because each is likely to make the other worse. If one is faced with the question of weakness, upon theoretical grounds the best food is protein, which has a tendency to diminish acidosis, and is the most strengthening food. Often a patient retains strength if given enough protein, and his diabetes improves through burning off body fat. Infection does not necessarily rule out expectant treatment, as our first case of carbuncle showed. The patient had been treated by an eminent surgeon, who advised immediate operation. We asked for twenty-four hours' delay. The toxic condition became no worse, and upon further delay operation was not necessary, and the patient became sugar-free. Much of this subject remains to be worked up by the competent internist in conjunction with the careful surgeon.

In regard to Dr. Tyson's question, I think, other things being equal, that the duration of the diabetes has a great influence upon the course

of treatment; the earliest case is the easiest to treat. The young cases seen early may do well. The young, severe cases untreated die. The cases running along for perhaps ten to twelve years, when the patients look well nourished and the diabetes does not seem to be hurting them much, are the ones likely to give dangerous results on fasting. They are the very type in which fatal symptoms upon fasting are to be watched for at any time. Breaking the fast by a restricted diet—even a protein-fat diet—clears up the acutely threatening symptoms in such cases and a second fast a week or two later is well borne. Sometimes the long-standing cases are slow to recover tolerance, and there may be required months of undernutrition and a worse state of health. It is an instance in which blind faith on the part of the patient in his medical adviser is essential. In the long run elderly patients do generally give better results than the younger ones.

Answering the other question, whether it makes any difference by what method the glycosuria is eliminated—if the blood be made normal, it probably makes no difference by what means this is done; but I am in accord with Dr. Taylor's conclusions on the basis of my experiments on dogs. Dogs brought to the point of diabetes, which will show heavy glycosuria on a bread diet, will live on a meat diet without any glycosuria. But if the dog be kept for months or years, I have seen in some cases that diabetes comes on; there is the spontaneous downward progress that has been talked about in human diabetes. The essential requirement is maintenance of life on a diet which will keep the patient sugar-free. The nutrition may have to be cut down to a little less than the ideal minimum. I am inclined to think that the undernutrition feature is more important than the elimination of any particular kind of food. A diet suitably restricted in protein and calories and containing carbohydrate in quantity safely below the tolerance, so as to keep glycosuria continuously absent, is probably the most satisfactory that can be given.

EPIDEMIC POLIOMYELITIS: ITS NATURE AND MODE OF INFECTION[1]

By SIMON FLEXNER, M.D.
NEW YORK

The present time, while we are under the influence of the serious epidemic of the past summer and autumn, may be the most favorable time to impress certain essential facts regarding poliomyelitis upon the general medical profession.

Epidemic poliomyelitis, or infantile paralysis, as the disease is variously and on the whole erroneously called, is becoming increasingly familiar to us. Indeed, each year since 1906 the disease has prevailed in some part of this country in severe or even epidemic form. It is, of course, true that epidemics had occurred in this country before, but their relatively small size and their infrequency marked them off sharply from the epidemics prevailing since 1906. The recent experience America has been passing through is, however, not exceptional, since during the same period the disease has become more prevalent throughout the world—affecting the European countries, the West Indies, South America, the far East, Australia, etc. This very wide and general distribution of the disease can be attributed to the endemic focus in northern Europe, ever becoming more and more active, which culminated in the severe epidemic outbreak in Sweden of 1905. It was the epidemic of 1905 which was the immediate forerunner of the pandemical occurrences mentioned.

Unfortunately, very considerable uncertainty exists still regarding the character and nature of this epidemic disease. Because of its recent introduction into this country a large part of the medical profession has lacked familiarity with it. On the surface it is not so

[1] Read December 6, 1916.

easy to identify the severe forms of the epidemic disease with the occasional and sporadic instance of infantile paralysis arising in every considerable community, and yet essentially they are one and the same disease—that is, they are due to a common etiological agent, in the same manner as the sporadic instances and epidemics of cerebrospinal meningitis are both caused by the meningococcus.

Considerable difficulty and confusion have been introduced into the subject by the common names for the epidemic disease. As is usual, names express conceptions, and in this case the names chance to be misapplied. So long as the disease is conceived of as one attended by paralysis, which in turn is due to lesions of the gray matter of the spinal cord (or brain), a large number of cases are excluded altogether. Because we have now learned, thanks especially to Wickman, who studied the Swedish epidemic of 1905, that many cases of the disease, perhaps the majority, in epidemic times are not associated with any paralysis whatever. I shall not elaborate this point; I introduce it merely to emphasize its importance in respect to the public health control of the disease. Others who follow will doubtless refer to this same point. But I wish to leave you under no doubt that the disease we are considering appears in a variety of clinical forms, some of which are very slight and trifling and others of the profoundest severity.

If I should be called on to define epidemic poliomyelitis—so-called—I should call it an acute infectious and communicable disease, attended sometimes, but by no means always, with involvement of the central nervous organs, as a result of which incident paralysis often results. The parts of the central nervous organs most frequently involved are the meninges, with which may be and often is associated injury to the gray matter of the spinal cord and brain leading to muscular paralysis.

In its essential nature the disease is an infection. We now know, thanks especially to the employment of monkeys for inoculation, that the microörganism causing it is very minute, filterable, indeed, but probably not invisible. That is, by particular cultivation methods a minute anaërobic microörganism has been secured which fulfils Koch's law of causation. But I feel that it is better to wait

until this experimental work has been confirmed in other countries in which epidemic poliomyelitis occurs before proclaiming the micro-organism as the established cause of the disease.

But what is of first importance is the discovery of the manner in which the microbic cause of poliomyelitis enters and leaves the body, because the mode of infection so largely controls the methods of prevention to be taken.

Tests carried out on monkeys have proved beyond doubt that the virus of poliomyelitis, so-called, exists regularly upon the mucous surfaces of the nose and throat and often of the intestine, with the discharges of which it may gain access to external nature. It has also been traced on the upper respiratory mucous membrane of healthy persons who may act as carriers, and has been found in several instances in the tonsils removed by operation several months after recovery from the acute disease, so that the existence of so-called chronic carriers has also been indicated.

In spite of doubts and disputes concerning the mode of infection, this much we know positively, and hence should take sanitary measures accordingly: the infectious agent can and does enter and leave the body by the upper respiratory mucous membrane, and may at least leave the body with the intestinal discharges. Whether there are still other avenues of infection we do not know. Neither do we yet understand the tendency of the disease to reach its height in the late summer and early autumn months. But in attempting to interpret that phenomenon we must still keep in mind that increasing intensive study is showing more and more that cases of the disease extend throughout the winter, and in two instances at least midwinter epidemics occurred in Sweden and Norway.

Under the circumstances the disease is to be regarded as communicable from person to person—by the infected sick in any of the many forms in which it appears and by healthy persons who may be contaminated by the sick. It is, of course, not possible to control by isolation all exposed persons. But a large part of the problem of the public control of the disease rests on early and accurate diagnosis, and the reduction of exposure of persons who may become potential healthy carriers of the agent of infection.

What is greatly to be desired is a simple, readily applied biological test which would give practically unmistakable answer as to whether poliomyelitis was present or not. Such a test has not yet been devised. We cannot use the cultures for this purpose because they are too difficult to obtain; we can, however, use lumbar puncture, which in 90 per cent. or more of instances yields a definite result. In general, it may be stated that, irrespective of the severity of the symptoms, lumbar puncture yields in cases of poliomyelitis a fluid, usually clear, but showing either morphological or chemical changes, or both these changes simultaneously. The mononuclear cells tend to be increased and globulin often is present. These changes in the cerebrospinal fluid, especially during periods of epidemic, should be regarded as presumptive evidences of poliomyelitic infection and measures of public protection taken accordingly. Moreover, as beginnings are being made in respect to a specific form of treatment, the employment of lumbar puncture and study of the changes in the cerebrospinal fluid are affording the basis for the application of treatment at the more favorable periods, before contingent paralysis has appeared, which may often determine whether the treatment will be effective or not.

I must ask your indulgence for presenting a paper merely in outline. If the points presented impress you at times as being perhaps aphoristic, may I remind you that there exists competent, as I believe, clinical and experimental data for them all.

SOME PRACTICAL CONSIDERATIONS IN THE ADMINISTRATIVE CONTROL OF EPIDEMIC POLIOMYELITIS[1]

By HAVEN EMERSON, A.M., M.D.
NEW YORK CITY

There are obvious practical limitations to the application of scientific laboratory and clinical knowledge in the public control of communicable disease. Furthermore, when the means of transmission, of immunization, and of detection of carriers of a disease are lacking the resources of the public health officer are sadly restricted. In view of the keen public and professional interest in the recent severe epidemic of poliomyelitis and the advantages which should accrue from a frank admission of the accepted facts, I venture to present the following statement of the situation as it must face professional advisers of public policies, so far as this particular disease is concerned.

The use of popular educational publicity will go far to teach parents the necessity of obtaining competent medical advice immediately upon the appearance of fever, pain, digestive disturbance, or acute symptoms of any kind in little children. Such advice is applicable at all times, but is listened to with respect only when the public is interested or is aroused to a particular danger. The benefit of following such advice is seen at once in the earlier home recognition of the common infections of childhood, a quicker appeal to the family physician, and in the more prompt and general reporting of communicable diseases of children to the health authorities. To the adoption of such advice we may attribute the decided reduction of the infectious diseases (pertussis, measles,

[1] Read December 6, 1916.

diphtheria, scarlet fever, and infantile diarrhea) during the past summer.

The health officer can, with propriety, warn parents against allowing the indiscriminate association of children, and particularly against contact with children in whose families there are acute illnesses of any kind. As a type of simple, direct, practical advice the following extract from a circular of information to the public will serve for issue through the field force and the newspapers:

Information for the Public.[1]

"Infantile paralysis (poliomyelitis) is a catching disease. How it is spread is not yet definitely known. In most cases the disease is probably taken directly from a sick person, but it may be spread indirectly through a third person who has been taking care of the patient or through children who have been living in the same household.

The early symptoms are usually fever, weakness, fretfulness or irritability, and vomiting. There may or may not be acute pain at this time. Later there is pain in the neck, back, arms, or legs, with great weakness. If paralysis is to occur it usually appears from the second to the fifth day after the sickness begins. Many cases do not go on to paralysis.

The germ of the disease is present in discharges from the nose, throat, and bowels of those ill with infantile paralysis even in the cases that do not go on to paralysis. It may also be present in the nose and throat of healthy children from the same family. Do not let your children play with children who have just been sick or who have or recently have had colds, summer complaint, etc. For this reason children from a family in which there is a case of infantile paralysis are forbidden to leave their home. If you hear of their doing so report it at once to the Department of Health.

Persons over sixteen years of age, from families where there are cases of poliomyelitis, may continue at work unless their business has to do with the preparation or handling of food or drink for sale.

If you hear of a case in your neighborhood and the house is not placarded, notify the Department of Health.

How to Guard Against the Disease. In order to prevent the occurrence of this disease, parents should observe the following rules:

Keep your house or apartment absolutely clean.

[1] Extract from leaflet issued by the New York City Department of Health, July, 1916.

Go over all woodwork daily with a damp cloth.

Sweep floors only after they have been sprinkled with sawdust, old tea leaves, or bits of newspaper which have been thoroughly dampened. Never allow dry sweeping.

Screen your windows against flies, and kill all flies in the house.

Do not allow garbage to accumulate, and keep pail closely covered.

Do not allow refuse of any kind to remain in your rooms.

Kill all forms of vermin, such as bed-bugs, roaches, and body lice.

Pay special attention to bodily cleanliness. Give the children a bath every day and see that all clothing which comes into contact with the skin is clean.

Keep your children by themselves as much as possible. Do not allow them to visit moving picture shows or other places where children may gather.

Children should not be kept in the house; they should be out of doors as much as possible, but not in active contact with other children of the neighborhood. Do not take them on a street car, unless absolutely necessary, or shopping.

Do not allow your children to be kissed.

It is perfectly safe to let your children go to the parks and playgrounds if only two or three of them play together; they should not play in large groups, and you should not let them come into contact with children from other parts of the city.

Remember that children need fresh air in the summertime, and outdoor life is one of the best ways to avoid disease.

If there is a public shower bath in a school in your vicinity, send the older children there every day for a shower bath. This is perfectly safe and will help keep them in good health.

Give your children plain, wholesome food, including plenty of milk and vegetables.

Keep the milk clean, covered, and cold. Do not allow the milk or any other food to be exposed where flies may alight upon it.

Wash well all food that is to be eaten raw.

In Case of Sickness. Remember that during the hot weather children are apt to have stomach and bowel troubles. If your child is taken sick with loose movements of the bowels, or with vomiting, do not at once fear that it must be infantile paralysis; it may be simply digestive disturbance. Give the child a tablespoonful of castor oil and plenty of cool water to drink, and *send for the doctor at once.*

If you cannot afford a doctor's services, telephone the Department of Health and one will be sent free of charge.

If a doctor or nurse from the Department of Health visits your

home, give them all the information you can. They are sent to show you how to keep your children well.

Do not give your children patent medicines or buy charms of any kind to ward off the disease. *The best preventive is cleanliness and strict observance of the rules that have been given.*"

And when the advice has been given, the most unexpected and extravagant applications of it may ensue. Many a family of children was housed for weeks, often in tight-shut rooms, even in July and August, the children's pale faces pressed against the window panes, mute evidence of their unreasonable imprisonment.

The mother whose neighbor's children across the tenement hallway are attacked with poliomyelitis, one of the four dying of the disease and another a permanent cripple, is not easily persuaded that the warning of the health officer in papers, circulars, and by the visit of the nurse does not logically demand home isolation of the well during the epidemic. When the visiting nurse is met by a door slammed in her face, where previously welcomed as the family's guardian and friend, we see how the laity reacts to the warning against contacts, the nurse being supposed to be constantly carrying disease from family to family. In the main, however, such educational propaganda is valuable but beyond this there is to my knowledge no warning or advice which can fairly be considered to produce any tangible results before or during an epidemic of poliomyelitis. There are sound reasons for advising the screening of living and eating premises against insects. There is always advantage in maintaining the person, the home, and the public highway in cleanly condition. Cooked food is safer than raw food for children in summer.

Up to the present moment there is no practical experience which can be claimed to show that environment, *i. e.*, housing, street dust, sewage, garbage, water, food supply, domestic or wild animals or insects, whether blood-sucking or not, has any relation to the origin or dissemination of poliomyelitis.

Aside, then, from the teaching of the principles of personal hygiene

and avoidance of fatigue, the need of obtaining medical services in the presence of sickness and the probable advantage of avoiding contact between children during an epidemic, the hands of the health officer are tied by his ignorance of the means of transmission and the identity of carriers who do not show evidence of sickness.

When a case of sickness occurs the difficulties do not diminish. Diagnosis cannot be said to be either easily or positively made unless there is a paralysis present at some time. The increasing probability that there are as many non-paralytic as paralytic cases does not satisfy the parents of children, who naturally look for some positive proof, specific for the disease when it is diagnosed in a non-paralytic form, before willingly relinquishing their child to hospitalization or submitting to a quarantine.

We have no specific diagnostic test, though the spinal fluid considered in connection with a good history and definite clinical symptoms will, during an epidemic, even in the absence of paralysis, justify a positive diagnosis.

The discordant opinions of other physicians, whose services are commonly obtained by a resentful family when report of a case of poliomyelitis is made by the regular physician in attendance, bring the official diagnosticians of the Health Department into difficult relations with their fellow-practitioners, and not uncommonly result in feelings of resentment on the part of the family, which in one instance led to the institution of habeas corpus proceedings.

In a disease so little seen or recognized in the past in general practice it is not uncommon to lose that agreement and approval among physicians without which it is obviously impossible to obtain public compliance with restrictions or professional coöperation with health officers so necessary for early and universal notification.

In the early days of an epidemic inevitably the diagnosticians of the Department of Health see more cases in a very short time than do other physicians, and if thoroughly trained under capable supervision they rapidly become experts to a degree which temporarily at least makes them appear critical of their fellow practitioners.

This difficulty is readily met by organizing public medical clinics and lectures at various points throughout the city where pediatrists,

neurologists, and Health Department diagnosticians may give personal instruction in the early and differential diagnosis of poliomyelitis.

The prompt issue of leaflets of information to physicians, early in the epidemic, will enlist their coöperation and give the essential facts in a form for ready reference. Such a circular as the following proved a helpful starting-point with many for a more thorough understanding of the disease with which, at least in its epidemic manifestations, few were at all familiar.

Information for Physicians.[1]

"*Early Diagnosis.* The attention of physicians is called to the necessity of an early diagnosis of all cases of poliomyelitis. Early recognition and strict quarantine are the chief weapons against the disease.

Reporting of Cases. All suspicious cases must be at once reported to the Department of Health by telephone, to be followed within twenty-four hours by a written report. The ability of the Department of Health to limit the spread of the infection depends upon the immediate reporting of every suspicious case.

Age of Persons Affected. It should be remembered that this disease may occur at all ages, although the great majority of the cases are found in children between the ages of one and five years.

Type of Disease. Peabody, Draper, and Dochez, of the Rockefeller Institute, give the following classification of the disease:

1. The non-paralytic or so-called abortive cases.
2. The cerebral group, with spastic paralysis.
3. The bulbospinal group.

Methods of Infection. The experiments of Landsteiner and Popper in Germany; Kling, Pettersson and Wernstedt in Sweden, and of Flexner and Noguchi in this country have proved that the disease is transmitted by the secretions of the nose and mouth and the bowel discharges of an infected person. The infection is transmitted through the mouth, tonsils, and nasal mucous membrane.

Contacts and Carriers. It must be remembered that while the transmission of the *disease* from a patient to other members of the same family is not usual, transmission of the *virus* is common. Experience warrants the assumption that in addition to direct contact the disease is spread by carriers, usually children, who are themselves immune but who harbor the infective material in their nasal or mouth secretions.

[1] Extract from a leaflet issued by the New York City Department of Health, July, 1916.

Symptoms. Early symptoms to be regarded as suspicious are: Fever, vomiting, slight diarrhea, listlessness, unusual fretfulness, and drowsiness. Later and more characteristic symptoms are: The appearance of weakness in any extremity, skin and muscular sensitiveness, spinal pain, especially on flexion, apparent or real rigidity of the neck muscles, Kernig's and MacEwen's signs.

Course and Duration of Disease. Paralysis appears usually before the sixth day of the illness; it may occur as early as the first day. Other symptoms, except spinal and muscular pain and rigidity and skin sensitiveness, rarely persist.

Non-paralytic or So-called Abortive Cases. Non-paralytic or so-called abortive cases are very frequent. In some epidemics they constitute from 25 to 50 per cent. of the diagnosed cases. The children have the early symptoms just mentioned, perhaps also the muscular tenderness and spinal pain. If carefully observed it may be noticed that they develop a paralysis of one or more groups of muscles, but that instead of the paralysis continuing it all disappears within a few hours. It is obvious that the recognition of such cases is of extreme importance in controlling the spread of the disease. The diagnosis of such cases is greatly facilitated by an examination of cerebrospinal fluid obtained through lumbar puncture.

General Care of Patient. Complete rest is of the utmost importance for either paralyzed or weak muscles for the first five or six weeks. Every effort must be taken to make this rest complete. The limb must not be allowed to drag on a paralyzed muscle. It should be supported by pillows or pads or bandages. There seems to be a greater tendency to atrophy if casts are used. A dropped foot may be supported by a sandbag or pillow; small rolls placed under the knee often hold the leg in a more comfortable position. The weight of the clothing should be kept off the legs by hoops or other device. If the head is somewhat retracted and the patient desires to lie on his back, he may sometimes be made more comfortable by a small pillow placed under the shoulders, allowing the head to fall back. The value of electricity for treatment in the first six weeks is very doubtful. In many instances it may do harm. Massage or passive movements should not be begun for at least five or six weeks and then should be used with great care. In cases that show a tendency to clear up rapidly the child should be kept in bed for some time after the ability to use the muscles returns. It should never be encouraged to try to stand or to use the muscles otherwise until a considerable time has passed.

Period of Incubation and Duration of Disease. The incubation period has been officially set at two weeks. Non-immune, infected

persons usually manifest symptoms of the disease in from five to ten days after exposure. The average period of incubation is seven days. The early symptoms, noted above, usually last from one to seven days. Quarantine of the patient will be maintained for a period of at least eight weeks.

Prevention of Spread of Infection. 1. The children from an infected family will be confined to the house. (See "Quarantine.")

2. During the continuance of an epidemic of poliomyelitis children should not be allowed to congregate in public places.

3. Fresh-air outings or vacation camps are allowed, if kept under competent medical supervision, with an adequate physical examination of each child before enrolment and the exclusion of any child from an infected family.

4. Absolute cleanliness of all homes is essential; such cleanliness should include:

(*a*) Screens in all windows.

(*b*) Flies kept out of all rooms.

(*c*) Thorough cleanliness of all floors, woodwork, bedding, and clothing.

(*d*) Avoidance of dust (all sweeping should be done after the floors have been sprinkled with sawdust, bits of newspaper, or tea leaves, all thoroughly moistened).

(*e*) Garbage cans kept closely covered and washed out in hot soapsuds after they have been emptied.

(*f*) No refuse, either of food or other waste, allowed to accumulate.

5. Personal habits of cleanliness are essential; the hands should be washed before each meal, after each visit to the toilet, and before going to bed. Children should be warned about putting the fingers into the mouth or nostrils.

6. When sneezing or coughing, a handkerchief should be held over the mouth. Kissing of children is also a dangerous practice and should be avoided.

Procedure to be Followed in Each Case. 1. Isolation of patient: Complete isolation of the patient must be maintained until terminated by order of the Department of Health.

2. A separate room must be provided for the patient. No one must be allowed in this room except the attending physician, the nurse, and the representative of the Department of Health.

3. Care of patient's room and surroundings: (*a*) All rugs, carpets, draperies, and unnecessary furniture must be removed before the patient is placed in the room.

(*b*) All windows must be screened.

(*c*) The sick room must be kept well aired at all times.

(*d*) The woodwork must be wiped daily with damp cloths. Under no circumstances must the floor be swept when it is dry. It should be sprinkled with sawdust, bits of newspaper or tea leaves, all thoroughly moistened and then carefully swept so that no dust may arise.

(*e*) Toys and books used by the patient must be destroyed by burning after recovery or death.

(*f*) Household pets must not be allowed in the room.

4. Care of bedding: All cloths, bed-linen, and personal clothing which have come into contact in any way with the patient must immediately be immersed in a 5 per cent. solution of carbolic acid and allowed to soak for three hours. They may then be removed from the room and must be boiled in water or soapsuds for fifteen minutes.

5. Care of discharges from body: A sufficient supply of gauze or clean linen or cotton cloth must be provided and all discharges from the nose and mouth of the patient received on these cloths. After use they must be immediately burned or boiled. Bowel discharges and urine must be covered at once with chloride of lime and then disposed of by emptying into a water closet.

6. Care of utensils used by patient: Plates, cups, glasses, knives, forks, spoons and other utensils used by the patient must be kept for exclusive use and under no circumstances removed from the room or mixed with similar utensils used by others. They must be washed in the room in hot soapsuds and then rinsed in boiling water. After use the soapsuds and water must be thrown into the water-closet.

7. Nurse: A trained nurse or competent attendant must be in sole attendance upon the patient. She must not be allowed to mingle with the rest of the family but must be isolated with the patient. The hands of the nurse must be carefully washed in hot soapsuds after each contact with the patient and before eating.

8. Termination of case: After the case has been ordered terminated by the Department of Health the following procedure must be followed:

(*a*) The entire body of the patient must be bathed and the hair washed with hot soapsuds. The patient should then be dressed in clean clothes (which have not been in the sick room during the illness) and removed from the room.

(*b*) The nurse should also take a bath, wash her hair, and put on clean clothes before mingling with the family or other people.

Action Taken by the Department of Health in Each Case. Placarding: Every house will be placarded without exception. In private

houses one placard is placed on the street front (outside of house), and one placard is placed on the door entering the room patient is in. In tenements three placards are affixed, one on street door, one in entrance hall, and one on door of apartment. All placards are dated.

Quarantine: In all families where a case of poliomyelitis has occurred all other children under sixteen years except those who have had the disease are to be quarantined in the home until two weeks after the termination of the case by death, removal, or recovery.

The patient, whether at home or in hospital, must be quarantined for eight weeks from onset of disease.

Children under sixteen years of age who have been, but no longer are, exposed to infection will be quarantined for fourteen days.

Removal to hospital: No case is to be left at home unless the following conditions are complied with:

(*a*) There must be a private physician in attendance regularly.

(*b*) Persons attending patient must obey quarantine rules; must not do any housework, marketing, or leave premises.

(*c*) Patient and attendant must have separate rooms.

(*d*) All windows of rooms used by patient must be screened.

(*e*) The family must have a separate toilet for its exclusive use.

(*f*) Quarantine regulations must be strictly observed by patient and other children.

Deaths: In case of death prompt burial is required, the coffin must be sealed as in deaths from other contagious diseases, and the funeral must be strictly private. Church funerals are prohibited.

Spinal puncture: Physicians desiring the services of a consultant to perform lumbar puncture and report on the examination of spinal fluid should telephone to the Department of Health."

Once the diagnosis is accepted by family and physician, the next step is fairly clear. Contact is effectively prevented in hospitals and less consistently in the home even when there is unusually intelligent and favorable care. Removal of the sick child to a hospital, if accepted by parents, is a simple matter; if opposed, it may prove the irritant which sets a whole community against the simplest and gentlest measures of restraint. Anything which causes antagonism of the public to the policy of reporting and

removal to isolation hospitals, develops deception, hiding of cases, and such methods of obstruction as to frustrate to a great degree any approach to successful separation of the sick from the well. Once the case is under surveillance at home or in hospital the uncertainty as to a reasonable period of isolation becomes prominent. The parents and the press ask embarrassing questions. What is the duration of infectiousness? If six weeks is not certain is six months any more so? Why eight weeks in New York City and six weeks elsewhere throughout the country? Six weeks has been generally agreed to as a suitable isolation period dating from the day of onset, the only adequate basis for this being that, after six weeks, infectious material has rarely been obtained from artificially infected monkeys. We established the isolation period at eight weeks in New York City at the beginning of the epidemic, to impress the public with the seriousness of the danger and partly to obtain a greater percentage of voluntary hospital admissions. There is no reason to believe that eight weeks' isolation of infected individuals is of any more value than the more commonly accepted six weeks' period.

How long the incubation period? Is a child recovered from paralysis still infectious? How long before the disease expresses itself has a patient been infectious? Only provisional answers can be offered to any of these questions.

Whether or not hospitalization justifies itself as a sanitary measure to protect susceptibles against exposure to infected individuals, it has resulted in definite and marked improvement over the usual treatment given to the children of the poor in their homes during the acute stage of the disease and in the prevention of much avoidable subsequent deformity.

There are too many undetermined factors which may have played a part, to draw positive conclusions as to the effect of hospitalization upon the distribution of poliomyelitis in New York, but it is certainly suggestive that in the Borough of Manhattan where more than 90 per cent. of all reported cases were removed to hospitals, the case incidence of the disease was 0.94 per 1000, while in Brooklyn, where not over 50 per cent. of the cases were taken

from their homes for isolation purposes, the case incidence was 2.24 per 1000 of the population.

The unusual opportunity offered by the assembling of such a number of acute cases and holding them for a long isolation period should not be lost to the medical profession. Postgraduate clinical teaching should be carried on as systematically as the emergency conditions prevailing will permit.

More than 800 physicians visited the wards of the Willard Parker Hospital, where, for a considerable period we had under observation 2078 cases of the disease. During the epidemic we had a total of 5437 cases under treatment in the hospitals of the Department of Health.

Health officers owe it to the practitioner of medicine to see that every facility is given to study and observe all clinical phases of the disease, and to inform himself as to the possibilities of early and accurate diagnosis and promising or unprofitable methods of treatment.

It is impracticable to allow each patient to be treated by the family physician, and it is undesirable to allow more than the minimum of visits by family or friends. Two visits by members of the family in eight weeks was our rule, and in this way we avoided the development and spread among the patients of the other communicable diseases of children. Complete exclusion of the family would prevent public support of the policy of hospitalization.

Morphological, cultural, and biological tests for the presence of the infecting organism are not yet sufficiently precise or uniform to offer any service to the public health officer. The detection of carriers or of the infectious preparalytic or non-paralytic cases is thus impracticable.

As to general measures of personal restraint directed to the abatement or prevention of an epidemic we are even more at sea than in the field of individual quarantine. As a means of impressing upon the public the seriousness of the epidemic and the need of caution, various measures may be justified, among them the ban upon public assemblages of children within confined spaces,

except where medical or nursing supervision and identification of homes or addresses of children could be maintained.

Identification of travelers, especially of children, and their supervision at their place of arrival for the presumed incubation period of the disease is another reasonable means of controlling obvious transmission of the disease; but the incompleteness of this under modern conditions of traffic and in view of the probability of the presence of large numbers of healthy carriers, children and adults, makes its advisability very questionable.

The alternative, one may say, is the absolute quarantine against people coming from places where the disease is prevalent. I hardly need to recall the countless instances of inconvenience, hardship, yes real brutal inhumanity which resulted from the application of the general quarantine of counties, towns, and States against each other during the past summer. I do not know of any health officer who can or has maintained that such measures have had the slightest effect upon the incidence or spread of the disease in his jurisdiction, and I know that nothing has developed so many automobile detours, such ingenuity in the violation of the laws, and such whole-hearted disrespect for reasonable sanitary law and its enforcement.

General reporting by the health officer at the point of departure to the health officer at the place of destination of travelers under the age of sixteen years would probably accomplish all that can be expected. Modern methods of sanitary control are directed toward localizing the disease in the person of the sick individual. Interference with the traveling of healthy people cannot be legally justified, nor will it be cordially supported by the public until health officers can make good their ability to detect and prove the presence of the virus in any one other than those actually suffering or recently recovered from the disease, and in a way that will not materially hinder personal freedom.

In the case of diphtheria, typhoid, cholera, cerebrospinal meningitis, tuberculosis, and malaria carriers we can with but insignificant inconvenience to the individual protect the public by the use of simple and accurate methods of diagnosis. Furthermore, the

rapidity and irregularity of the spread of the disease to other communities develops amazing and laughable inconsistencies in any efforts at local quarantines, as, for example, was the case of quarantines enforced by various communities in New Jersey, Connecticut, and Long Island when the communities outside the city showed a case incidence in some instances from two to four times as high as that of New York City itself.

If it is admitted that consistent and scientific control of communicable diseases is properly directed to preventing the escape of infectious material from the immediate person or vicinity of the infected individual, and if we can believe the records which indicate that epidemics of poliomyelitis do not occur in schools and institutions as they do commonly in the case of other communicable diseases of childhood, then interference with attendance of children at school is hardly excusable except as a concession to the apprehension of parents. This applies with still greater force to the application of prohibitory regulations to institutions of learning for students of sixteen years or over. The experience in New York City in the public schools and in the City College and other colleges and trade schools fully supports this view.

It is to be deplored that many private schools and colleges adopted regulations calculated to protect their patrons, though with the reasons for their measures based more upon the fears of the parents than upon any probable protective results. The managers of schools and colleges, depending for their income and the support of benefactors upon the good-will of present or prospective students and their parents, may find it good policy to take extraordinary precautions to prevent the invasion of poliomyelitis with the assembling of students for the fall term. One of the results of such additional restrictions has been the development of a feeling often expressed that the children of the poor were but doubtfully protected by the public officers, while the rich received the benefit of a wiser and more conservative policy. Nothing in our experience with the 900,000 public school and parochial school children in New York City gives any support to this point of view.

Uniformity of action among school and college principals in September would have gone far to allay public alarm instead of permitting additional confusion in the public mind and inconveniencing to no good purpose many thousands of families.

Unless there are demonstrable results to be expected from the application of more drastic regulations, those adopted by qualified public health authorities should be accepted and followed by private governing bodies of schools, and colleges, with the approval and advice of their own medical consultants.

Just as interstate uniformity of procedure is advisable under Federal supervision, so intrastate regulations in the several towns, etc., should be as nearly identical as possible.

While awaiting the receipt of the essential information which will justify a logical and efficient administrative control of epidemic poliomyelitis, the health officer of any community can hardly do better than plan his campaign in matters of regulation and public education in accordance with the declaration of the American Public Health Association, which I take the liberty of quoting in full:

"The specific cause of poliomyelitis is a microörganism, a so-called virus, which may be positively identified at present only by its production of poliomyelitis in monkeys experimentally inoculated. Such experiments have shown this virus to be present not only in the nervous tissues and certain other organs of persons who have died of poliomyelitis, but also in the nose, mouth, and bowel discharges of patients suffering from the disease. It has been proved by similar experiments that healthy associates of poliomyelitis cases may harbor the virus in their noses and throats.

These experiments, together with the fact that monkeys have been infected by direct application of the virus to the mucous membrane of the nose and by feeding of the virus, are strong evidence that in nature infection may be directly spread from person to person.

Observations on the occurrence of the disease might seem at first thought to be inconsistent with this conception, since contact between recognized cases can seldom be traced. However, this may be adequately explained by the lack of means for detecting mild

non-paralytic cases, and by the belief that healthy carriers of the virus and undetected cases are considerably more numerous than frankly paralyzed cases.

Many facts, such as the seasonal incidence and rural prevalence of the disease, have seemed to indicate that some insect or animal host, as yet unrecognized, may be a necessary factor in the spread of poliomyelitis, but specific evidence to this effect is lacking, and the weight of present opinion inclines to the view that poliomyelitis is exclusively a human disease and is spread by personal contact, whatever other causes may be found to contribute to its spread. In personal contact we mean to include all the usual opportunities, direct or indirect, immediate or intermediate, for the transference of body discharges from person to person, having in mind as a possibility that the infection may occur through contaminated food.

The incubation period has not been definitely established in human beings. The information at hand indicates that it is less than two weeks, and probably in the great majority of cases, between three and eight days.

If the foregoing conception of the disease is correct, it is obvious that effective preventive measures approaching complete control are impracticable, because isolation of recognized cases of the disease and restraint upon their immediate associates must fail to prevent the spread of infection by unrecognized cases and carriers.

These difficulties would appear to be inherent in the nature of the disease. Nevertheless, we may hope for the development of more thorough knowledge which will permit of more effective control of the disease than is now practicable. Of first importance is the more general recognition by practitioners of non-paralytic cases through clinical observation and laboratory procedures.

Lumbar puncture has been shown to offer valuable aid in diagnosis and a more general use of this test is to be encouraged, since it not only facilitates accurate and early diagnosis, but in many cases affords symptomatic relief as a therapeutic procedure. Without undertaking to predict the future progress of research, we may hope for certain possible developments which may afford far more effective control of the disease, with substantial relief from many inconveniences at present inevitable. Among these possibilities we would include: a practical test for the detection of all clinical types and carriers; a simple and reliable test for distinguishing between susceptible and insusceptible persons; and means of conferring artificial immunity against poliomyelitis.

At present our information demands the employment of the following administrative procedures in attempting to control the disease:

1. The requirement that all recognized and suspected cases be promptly reported.

2. Isolation of patients in screened premises. The duration of infectivity being unknown the period of isolation must necessarily be arbitrary. Six weeks has been recommended by the Conference of State and Territorial Health Officers with the Surgeon-General of the Public Health Service as sufficient, and this period has been generally accepted throughout the United States.

3. Disinfection of all body discharges.

4. Restriction of the movements of intimate associates of the patient so far as practicable. This should include at least exclusion of the children of the family from schools and other gatherings.

5. Protection of children so far as possible from contact with other children or with the general public during epidemics.

6. Observation of contacts for two weeks after the last exposure.

There is no specific treatment of established value in poliomyelitis. During the persistence of the acute symptoms of the disease the important principles of treatment are rest in bed, symptomatic relief, and passive support for the prevention of deformities. Active measures during this stage are not only useless but are apt to cause serious and often permanent injury. Hospitalization of patients when possible is to be encouraged. The best chances of recovery from residual paralysis demand skilful after-care, often long continued, and always under the direction of a physician familiar with the neurological and orthopedic principles of treatment. The provision of such after-care often becomes a community problem, demanding the coöperation of all available agencies, social and professional."

As a matter of historical interest and to present the result of the experience of the Department of Health of the City of New York, I append the following text of information for field workers which describes the procedure in the various phases of the work undertaken in the effort to check the spread of the disease last summer:

"1. General Information. *Incubation Period.* The incubation period of the disease and the quarantine period of children under sixteen years of age who have been, but no longer are, exposed to infection has been set at fourteen days.

Quarantine. In all families where a case of poliomyelitis has occurred, all the children under sixteen years (except those who have had the disease) are quarantined in the home until two weeks after the termination of the case by death, removal, or recovery. The patient, whether at home or in hospital, is quarantined for eight weeks from the date of onset of the disease. No case in hospital may return home until quarantine is ended.

Placards. All premises where a case of poliomyelitis occurs are placarded, the only exceptions being hotels and boarding houses, which are not placarded provided patient is at once removed to hospital, the room or rooms immediately disinfected, and no quarantined children remain on the premises. In private houses one placard is placed on the street wall of the house and one on the door entering room the patient occupies. In apartment and tenement houses three placards are posted—one on the street wall, one on the wall of the entrance hall, and one on the door of the apartment. All placards must be dated and initialed.

Removal to Hospital. No case may be left at home unless the following conditions are complied with:

(*a*) There must be a physician in daily attendance.

(*b*) The patient must have a special attendant who must obey quarantine regulations and must not do any housework, marketing or perform any household duties for other members of the family. He or she can, however, leave the house provided the necessary precautions as to personal disinfection, etc., are observed, but should avoid all children.

(*c*) The patient and the attendant must have a room, or rooms, separate from the rooms of others in the family.

(*d*) All the windows of this room must be screened and all flies in the room killed.

(*e*) The family must have a separate toilet for its exclusive use.

(*f*) Quarantine regulations must be strictly observed by the patient and the other children of the family, if any. When the disease occurs in the premises of families of food handlers the employment of such person or persons at this occupation is forbidden, unless they occupy entirely separate apartments for a period of two weeks after the removal, recovery, or death of the patient.

(*g*) Disinfection and renovation: The personal and bed-linen of the patient must be properly disinfected and, after removal, recovery or death of the patient, complete renovation of the room or rooms occupied by the patient and attendant is required.

2. Duties of Inspectors. Cases are reported by physicians, nurses, social workers, and other citizens, and all are visited at once by inspectors, even those reported by physicians, with request that they be admitted to hospital. Attending physicians to Department Hospitals may admit cases direct, without inspector's visits.

The janitor or his representative must be seen in every instance and notified that he or she will be held personally responsible by the Department for keeping quarantined children in the family premises and seeing that placards are not removed or defaced.

If the inspector makes or confirms the diagnosis of poliomyelitis, the Borough Office of the Department is notified and by it the ambulance is summoned, if removal is indicated. In every case the inspector leaves the hospital admission slip, properly and fully filled out. When case is left at home, inspector must give full instructions to family.

All cases of questionable diagnosis must be seen at once in consultation with the Borough or Chief Diagnostician, and whenever it is required, spinal puncture will be made and laboratory report submitted by the staff of the Research Laboratory. Cases with positive laboratory findings will be considered as poliomyelitis, regardless of clinical signs. A full history must be recorded on a special card for each assignment covered by inspectors.

3. Duties of Nurses. Nurses will visit every case reported, to instruct the family regarding quarantine, and every other family in the house:

(*a*) That there is a case of this disease in the house.

(*b*) That the other children of the family in which the disease has occurred will be quarantined, and that should they fail to observe quarantine, that fact should be immediately reported to the Department of Health, when steps will be taken to enforce quarantine by a summons to Court.

(*c*) Regarding home cleanliness, personal hygiene, the danger of infection by flies, and other general measures which should be taken to prevent infection.

(*d*) To report at once to the Department any cases of suspicious illness of children, or any case of poliomyelitis, especially if there is no physician in attendance.

A current history must be kept by the nurse for every case, giving dates of visits, action taken, and date and mode of termination.

Nurses must see the janitor or his representative on first visit, and repeat the instructions given by the inspector.

Patients remaining at home and families with quarantined

children are visited daily by the nurse or patrolman for the maintenance of quarantine, and oftener if necessary. After removal, recovery or death of the patient, nurses issue renovation notices, following these up by visits until complied with.

4. Duties of Sanitary Police. These officers visit frequently—daily if necessary—quarantined premises, to enforce quarantine of patient and other children in the family, and to affix or replace placards. They serve summonses when quarantine regulations are violated and appear in Court.

5. Ambulance Surgeons. All cases ordered removed to hospital must be removed by the ambulance surgeon without question, with the following exceptions, in each of which the ambulance surgeon must first obtain telephone authorization from the Resident Physician of his hospital to leave the case at home:

(*a*) When removal would endanger life of child (bulbar cases).

(*b*) When family physician can show that requirements will be met at once (or within twelve hours).

Doubtful and mixed infection cases must be removed by themselves in a separate ambulance.

In every case ambulance surgeons must leave a card with parents, giving name and address of hospital to which patient is taken. If inspector has not left admission slip, surgeon must make out same.

6. Visitors to Hospitals. Each case may be visited twice during its stay in the hospital by a parent or guardian. If child is critically ill the guardian or parent will be notified and will be permitted to visit daily while child is dangerously ill. Information relative to condition is given out at the Information Desk in each hospital, or by telephone in response to telephone inquiry from the parent or guardian.

7. Certificates for Children Leaving the City. The Department of Health of New York City does not require certificates of anyone leaving or entering the city. It issues certificates only as a convenience and aid to persons leaving the city. None are issued to persons passing through the city.

Such certificates state that the persons or family therein named have not resided in a house where a case of poliomyelitis has occurred. The applicant must sign a request for the certificate. They are refused to persons who live in a house where a case of infantile paralysis has occurred or who present symptoms of the said disease.

The certificates are good only until midnight of the following day, except when issued on a Saturaday or on the day preceding

a holiday, when they are good until midnight of the second following day.

Persons Leaving New York State. Officers of the U. S. Public Health Service, stationed at transportation terminals, require the above certificates before they will permit children under fifteen years of age, resident in New York City, traveling to points outside the State of New York, to leave the city. The original applicant must again sign the certificate in the presence of the Federal Health Officer. Federal Health Officers do not require certificates of any adults.

Persons Going to Points within New York State. Residents of New York City, adults or children, traveling to points within New York State, who present certificates of good health from their family physicians, may also obtain the above certificates from the Department of Health. If no physician's certificate of good health is presented, applicants will be examined by a physician and their freedom from symptoms of poliomyelitis certified; in this case all children must be brought to the proper office of the Department.

8. Return of Cases of Poliomyelitis to New York City. Cases of poliomyelitis occurring in residents of New York City who are temporarily residing outside the city, and developing within two weeks of the time of leaving the city, will be permitted to return, provided (*a*) a private conveyance (private car, private automobile, carriage or ambulance) is used, and (*b*) the patient goes direct to a hospital authorized by the Department of Health to care for cases of poliomyelitis.

Cases in which the onset of the disease occurs two weeks or more after leaving the city, may not return to New York City until eight weeks from the date of onset of the disease. But in special cases, where proper medical, surgical and nursing care is not obtainable, patients may be brought back to the city in a private conveyance, providing they go directly to a private room in a private hospital authorized by the Department of Health to receive cases of poliomyelitis.

9. Return of Children Who have been Exposed to Poliomyelitis to New York City. Children under sixteen outside of New York City who have been exposed to infection with poliomyelitis within two weeks, may return to the city under the following conditions:

They must come by private conveyance and must go direct to their homes.

Advance notice must be sent, and authorization obtained, by

telephone, by the local Health Officer. Such notice must give the name and age of each child, together with the identified address, including the floor, and the latest date of exposure to infection, and must be followed immediately by a written notice.

Such children will be promptly visited at their homes by a representative of the Department of Health, and instructed as to nature and duration of quarantine. They must not leave the premises until two weeks have elapsed from the date of last exposure to infection.

The premises are not placarded, but the children are visited at regular intervals, and should quarantine be violated the parents or guardians are summoned to Court and fined."

DISCUSSION ON PAPERS BY DRS. FLEXNER AND EMERSON

Dr. C. H. Lavinder (of the U. S. Public Health Service): I wish to express my pleasure at being present on this occasion to hear these two interesting papers on a subject of so much present importance to us all.

It was my privilege during the past summer to be associated, in a way, with Dr. Emerson during the epidemic in New York City. I know he can speak from experience, and doubtless with feeling, as well.

Dr. Flexner's paper, being on the mode of transmission, is of special interest and importance to every public health official.

Naturally, the object of the health officer in dealing with infectious diseases is prevention or restriction of spread. To him, therefore, the mode of transmission is of paramount importance. Without full and accurate knowledge of this feature of the disease, all his efforts are seriously handicapped. In poliomyelitis not only is our knowledge of the mode of transmission imperfect, but we also lack any means of accurate diagnosis and we possess only incomplete information as to the incubation period of the disease. There are three fundamental things in the epidemiology of an infectious disease, more especially when confronted, as we are in poliomyelitis, both with "missed" or "abortive" cases and with healthy carriers.

Studies as to the mode of transmission can be made by two methods: (1) laboratory research work, and (2) epidemiological field studies. The evidence so gathered does not possess the same value. The evidence from field studies is often fallible while that from the laboratory is much more dependable. The epidemiological method, however, is of great value provided its limitations are well understood. The two methods supplement each other, and when the truth is known their results should be in full accord.

With these difficulties in mind, the United States Public Health Service in the recent epidemic undertook certain studies. In the accomplishment of this work I wish here to acknowledge the hearty coöperation which we have received everywhere from local health authorities. We desired to take an extensive view of the New York epidemic, taking New York City as its center and studying the development of the disease in adjacent States. In addition to this, in order to obtain a cross-section of the epidemic, we undertook the intensive study of cases in several places: the total cases so studied numbered between seven and eight hundred. The time and labor involved in studies of this character are great and the work of compiling, tabulating, and digesting our data is not yet completed.

The officers associated with me, who went into the field to make studies, were in the beginning skeptical as to poliomyelitis being a contact disease. After some weeks of work, however, they almost unanimously became convinced of the contact idea, but could not give very conclusive reasons for this belief.

At this stage our studies do not justify perhaps any broad conclusions. From a preliminary survey of the results, however, there are many things which indicate that poliomyelitis is a contact infection. The matter may in a general way be briefly stated: In the first place it is undoubtedly true that doctors, nurses, and others in attendance upon the sick do at times develop the disease, although somewhat infrequently. Cases of the disease undoubtedly occur in groups, which is significant. When the disease invades a community previously free of it, it can very frequently be shown that the development of the infection occurred through contact by persons with another infected area. In this connection it is worthy of comment that the cases which seem so peculiarly isolated in a great many instances, upon investigation prove to be unfounded. A typical example of this was published recently in public health reports. A child on an island off the coast of California was reported to have typhoid fever. An officer was sent to investigate. He suspected poliomyelitis. The child was in the habit of making visits to a nearby village and had made such visit within ten days previous to being taken sick. Inquiry in this village at first developed nothing, but after persistent search a child was found paralyzed who had recently had a febrile illness. This proved to be a case of poliomyelitis, and two or three others were found. Further investigation disclosed the fact that these cases had all appeared subsequent to the advent of a family from New York City. A number of similar instances could easily be reported. Another factor which seemed quite evident from studying spot maps is that the disease followed travel routes. Moreover, it also seemed obvious that the disease spread in waves from a center outward. This was evident both in the chronology of the cases and the

incidence rates among the populations. We also observed that those parts of the population which escaped the disease many times were composed of persons who came less in contact with the general public than others; in New York City, for example, the children's institutions, which were under a modified quarantine and certain islands near the city. Furthermore, the acceptance of some mode of transmission other than contact would seem to lead us into even greater difficulties. In short, while the contact idea may be "hard to believe, it is still harder not to believe." Then, finally, the striking analogy which is shown between the epidemiology of cerebrospinal fever and poliomyelitis is a matter of consequence. This was, I believe, first pointed out by Dr. Flexner. In cerebrospinal fever we have quite accurate knowledge as to the mode of transmission and we know that we are dealing with a bacterial disease where the carrier plays an enormous part. In a recent discussion before the Royal Society of Medicine, Arkwright made this striking statement with regard to cerebrospinal fever. He said: "The number of carriers constitutes the mass of the epidemic and the real cases form only a small accidental minority." A statement of this character is certainly illuminating when we consider certain things which are observed in poliomyelitis.

When matters of the kind above stated are taken into consideration along with such laboratory evidence as we possess, to my mind they make a strong case for the idea that poliomyelitis is a disease spread by personal contact in its widest sense.

Of course, I know there are not a few serious objections and inconsistencies in this point of view. It is by no means easy to explain satisfactorily such things as peculiar seasonal incidence, age incidence, small incidence among general population, the fact that frank cases of the disease only infrequently produce secondary cases and so on. When we reach a fuller knowledge, however, many of these inconsistencies will doubtless disappear and harmony will prevail.

Of course, we shall doubtless learn many lessons from this epidemic. At present there seems to me to be two things at least worthy of consideration. The first of these is that poliomyelitis can be recognized much more readily by careful examination than was heretofore deemed possible, and if our clinical examination is supplemented by spinal puncture, our diagnostic ability is tremendously increased. Spinal puncture is a method which deserves much wider use in this disease. The New York State Health Department in this epidemic made wide use of it in some of their field work quite successfully.

Another important matter in this connection. The statement is often made that poliomyelitis is a relatively unimportant disease. I do not think that we can make such statements any longer. This disease has

been reportable in most States since 1909. In the registration area, which comprises now about 65 per cent. of our population, there were reported from 1909 to 1914 inclusive 5239 deaths from poliomyelitis. Roughly speaking, then, we might say that we have had in the United States from 1909 to 1915 perhaps 40,000 to 45,000 cases. This year we have certainly had in the United States not less than 35,000 cases, with a much higher mortality. This would certainly indicate that poliomyelitis is likely to prove now one of our important epidemic diseases of childhood.

Time does not permit any discussion of the position of the health officer in his attempts to control this infection. The difficulties are large, but it is imperative for us to do all that is possible with the knowledge that we possess. It would seem wise to be perfectly frank with the public, and then no one will expect the brilliant results which are impossible at the present time.

Dr. Wilmer Krusen (Director of Health, Philadelphia): We are deeply grateful to Dr. Flexner, Dr. Emerson, and Dr. Lavinder for coming to us with a report of their troubles in New York. We in the Department of Health in Philadelphia can sympathize with them. It is not my purpose to discuss this subject from the scientific stand-point but rather from the statistical and administrative. From June 1 to December 1 of this year the total number of reported cases in Philadelphia of anterior poliomyelitis was 997. As you well know, many abortive cases have not been reported. The diagnosis in those cases we have learned is difficult, possibly impossible. The total number of deaths was 297, a percentage of 29.2. Dr. Emerson has spoken of the benefit of Hospital treatment in these cases, and I want to present for your thoughtful consideration these facts: Of the 224 cases treated at home 125 died, a mortality of 53 per cent. Of the 763 patients treated in the Hospital 172 died, a mortality of only 22.5 per cent. This is an argument which we advance in favor of the hospital treatment of these cases. I will admit that many of the patients who died at home were in a very serious condition when the disease was determined. One thing which has interested me very much is that the density of population has no influence upon the number of cases in a given ward. For instance, the twenty-second ward, in Germantown, has a population of only ten per acre, yet there were 78 cases in that ward. The Third ward, one of the so-called slum districts, one of the most congested in the city, with a population of 210 per acre had only 10 cases. In the Thirty-ninth ward, with a population of 18 per acre, there were 63 cases; in the Forty-third ward, with a population of 46 per acre, 63 cases were reported; in the Second ward, with a population of 143 per acre, there were 55 cases; and in the First ward, with a population of 107 per acre,

there were 51 cases. The charts here presented show the dissemination of the cases, and you will notice that the Twenty-second, Forty-third, Twenty-ninth, Thirty-ninth, Second and First wards were the ones largely affected. Regarding the onset of the disease, 51.4 per cent. of the cases occurred in August. This I think corresponds with the incidence of the disease as mentioned by Dr. Emerson. September showed a percentage of 31.9; July, 8.9 per cent.; October, 7.7 per cent.; November, 1.5 per cent.; a gradual diminution to the present time. There was a popular belief at the beginning of the epidemic that the colored race was immune. In fact, I received a letter from a thoughtful old lady advising me to take the blood of colored children and inject it into the white for immunization. This belief, however, has not been borne out by actual experience, since we have had 23 cases of colored children with 8 deaths; 88 per cent. of the children affected with the disease have been under six years of age. One thought suggested to me of special significance by the chief of the Bureau of Health is that of the diminution in the average rainfall of the present year. Ordinarily, we have forty-two inches of rainfall in Philadephia annually. This year we have had a rainfall of only twenty-seven inches, an accumulated deficit of fifteen inches. Whether there is a relationship between the cleansing quanity of the storms and the occurrence of the disease is a matter I will leave to the laboratory men.

One matter of great practical interest is the after-treatment of these children who are usually of poor parentage. Many of them will need prolonged treatment. They will require braces and orthopedic appliances. This need has been taken up by the home Relief division of the Emergency Aid Society. They have raised a fund of money for the purpose of aiding these children whose parents are unable to purchase the necessary appliances. As you know, the various hospitals have opened wards for the after-treatment of these cases after they have left the Hospital for Contagious Diseases. Tonight we have only 15 cases remaining in the hospital, the smallest number since July 1.

What of the future? What is the prospect of an epidemic next year? We are hoping that we may not have an epidemic in Philadelphia next year, but the College of Physicians can help the Health Department of this city in its crusade for cleaner streets, for the elimination of the piggeries and all other nuisances, and in all work which will lead to a cleaner Philadelphia. We ask for your coöperation and support.

Dr. Theodore H. Weisenburg: My remarks have to do entirely with the clinical aspects of the disease. We had in Philadelphia an exceptional opportunity for studying infantile paralysis. There were in the Municipal Hospital about 800 cases, one-tenth of the number in New

York. This in comparison is not as large, yet it perhaps gave us a better opportunity for more intensive study. We not only observed the cases in the Municipal Hospital, but we also studied in many instances the localities and houses from which the patients came, and our investigations were concerned not only with the frank paralytic cases but also with the abortive types.

I started with the idea especially after reading Wickman's excellent work that there was no doubt whatsoever of the contagiousness of the disease. However, the more I saw of the disease the less and less was I of that opinion, and now I believe that the disease is not as contagious as either Drs. Flexner, Emerson, or Lavinder believe. I based my opinion altogether upon the clinical experience obtained in this epidemic. The best argument against contagiousness is to be found in the multiple instances of the disease occurring in the same family. It was our experience in Philadelphia that whenever more than one person in the family was stricken that the symptoms appeared within one or two days of each other, and in only one instance was the period of incubation longer than a week. Our experience with the abortive cases was similar. If the period of incubation is about seven days, the facts stated certainly argue against contagiousness, and point very strongly to the source of origin being identical.

Dr. Lavinder, as I understood him, stated that quite a number of persons coming in contact with the disease afterward developed it. This was not our experience. Of the large number of residents and nurses who came in contact with the sick children for quite a long period there was only one suspicious death in one of the resident physicians. There is, however, a well-grounded opinion that this physician died from tuberculous meningitis. Most of the nurses employed in the Municipal Hospital were young, as were also the residents. I have repeatedly seen nurses pick up children, kiss and fondle them, and I have also seen resident physicians go to a meal without cleansing their hands, yet none developed the disease. Besides that, when the schools were opened in Philadelphia in September, bringing together many children, it was to be expected that there would be an increase in the number of cases of poliomyelitis; yet just the opposite happened, the disease gradually diminished. Certainly, all these facts argue against personal contagion.

Dr. Emerson made an excellent suggestion regarding the postgraduate study of the disease in the various municipal hospitals. Should poliomyelitis reappear in Philadelphia, it should be the aim of those in charge to furnish the opportunity for every physician to study the disease. I quite agree with Dr. Emerson regarding what he terms the brutal quarantine in the different cities and States. With my small daughter, aged five years,

I had the unfortunate experience of motoring through Connecticut, New York, and Pennsylvania, and I can speak rather feelingly of the injudicious quarantine exercised by many communities through which I passed.

I am entirely opposed to interstate quarantine. First of all I do not see that any benefit is to be gained by arbitrarily quarantining one State against another, for to my mind nothing can be gained by keeping healthy children from their homes. I am, however, strongly in favor of isolation of the individual patient. Besides that, we know from our experience in this epidemic that the best therapeutic measure we can employ is lumbar puncture. In my experience this is practically the only measure that is of value, and the quicker it is done, of course, the better for the patient. The average physician outside of a large city, I am sorry to say, is afraid to perform lumbar puncture. Why should not a sick child, therefore, be given the advantage of such treatment, and if it cannot be done by the local physician, why should such a patient not be brought to the cities where such measures can be employed, and yet the quarantine absolutely forbids such a contingency.

Dr. Theodore Le Boutillier: After what we have heard this evening I feel that it is practically impossible to add to this discussion anything that is worth while. I would like, however, to say one or two words regarding what we considered the best method of procedure in treatment during the acute stage. Working with the cases as they came into the Philadelphia Hospital for Contagious Diseases, our first step was to do a lumbar puncture. That, as you have all heard, is not only one of the best methods of diagnosis, but is one of the best methods of treatment that we so far have. It relieves congestion, relieves pressure, and in many cases it will relieve pain to a very marked degree. Lumbar puncture to be efficient should be done frequently, that is, as each individual case demands it. This may be once every twenty-four hours for some days; it may be once a week, or once in two weeks, depending entirely upon the individual child. In a case studied by the physician in charge it is frequently very easy to tell when lumbar puncture should be done. The child complains of some pain, becomes restless, does not have a good appetite, and possibly has some headache. In 9 cases out of 10 lumbar puncture will relieve the condition. Of course, the fluid should be studied for the cell count. The protein and sugar content in our experience have not been of much diagnostic value. Children three and four months after the onset of the disease who have recurrence of pain, irritability, and restlessness will be readily relieved if lumbar puncture is again done. Such a case was seen three months after the original attack. Lumbar puncture had not been done until six weeks after the onset. This gave tremendous relief from pressure

symptoms. Six weeks later the symptoms returned in almost as severe form as before, when lumbar puncture was again done with complete relief of symptoms at that time. I believe that a case should be kept under observation for some time following their discharge from the hospital and watched for these returning symptoms. There was a boy, aged eighteen years, in the hospital who had been doing beautifully for weeks and who was to go to another hospital. The boy complained of intense pain in the elbow and general discomfort. Lumbar puncture was done at once and 35 c.c. of fluid removed. Within three hours the pain was entirely gone and the patient had better use of the elbow and arm than for a few days preceding.

Serum, I also believe, is helpful in many cases. The serum from the blood of patients who have had the disease some months or years previously I feel absolutely sure has saved the lives of some patients and improved very materially their condition. In other cases in which it has been given too late, or in insufficient quantity, or in which it has not contained the antibodies expected, the results have not been so good. We have had practically no bad results when the serum has been given. We could not obtain enough of the serum to give routinely in the quantities desired. We have used a combination of adrenalin chloride intraspinally and the serum intravenously. Our results have been better than when only one or the other was used.

There is in the study of poliomyelitis a tremendous field for laboratory work and for the clinician in finding a serum which will control a large percentage of these cases. I do hope that before the coming year is over such a serum may be produced which, if not as efficient as the diphtheria antitoxin, will at least be more helpful than anything so far discovered. There is a great opportunity for someone to discover such a serum and to use it.

I would emphasize the importance of the early diagnosis of the disease, the use of lumbar puncture not only as a diagnostic measure but a therapeutic, and the importance of not being afraid to employ this method frequently. It certainly has given me very great pleasure to listen to the papers of the evening.

Dr. G. G. Davis: Mr. President and Fellows of the College: The importance of the subject has been dwelt upon from the stand-point of the cause and spread of the disease, but in a less extent to the handling of it. This has largely to do with it as an acute infectious disease, but independently of that, it is important because it casts upon the community a large number of cripples with all the deleterious effects which follow.

This disease has found the profession unprepared and the public unpre-

pared. We who were familiar with the original type of the disease regarded it as an acute trouble for a few days with an immediate onset of flaccid paralysis and abrupt cessation of the acute symptoms. It therefore followed that the conditions being local in character the cases fell at once to the care of the orthopedic surgeons who treated all the paralytic cases. Orthopedics is a specialty, fortunately or unfortunately, and nowadays the time of our curriculum is so taken up by the wide fields of general medicine and surgery that orthopedics is restricted to a comparatively few, therefore this disease finds the practitioner unprepared to care for these cases.

When an emergency arises, there is a response from the community. How efficient this has been from the scientific stand-point of the cause of the disease and its early handling is evident from the brilliant array which has preceded me. I hope that the remainder of the profession may awake to its responsibility in regard to treatment.

The disease at present, as Dr. Flexner has said, is not as it has been heretofore. We no longer have a disease strictly limited to a certain period. We now have three instead of two stages. Instead of the acute onset of two or three days and the consequent paralysis, we have the acute onset, the convalescent and the chronic stages. Everyone will agree that the acute stage should be attended by the physician. We find, however, in this epidemic, even in the acute stage, meningitic symptoms, and we find to our surprise that spastic contractions are occurring early. My experience with the acute stage of the disease is not sufficiently large to say how early these contractions may occur, but certainly within a very few weeks. As the physician must be prepared to cope with certain local troubles early in the affection, he should have in consultation a person accustomed to guard against the oncoming deformity. Even inside of six weeks there ought to be some provision made in a certain number of cases against the contractures which will probably occur.

In the stage of convalescence after leaving the hospital, the family physician may be the attendant. He is confronted with a disease which even to the most experienced in its special character is baffling. There will probably be found a great number of these cases, carefully and tenderly cared for by the parents and by family physicians, but who do not know what the children require. They will be allowed to become permanent cripples when this might be prevented. It is unfortunate that our resources are so limited, but when a child is discharged from the hospital at the end of six weeks, three or four measures are at our command: Massage, the application of heat, the prevention of deformity by means of light splints, the use of electricity and exercises. All of these are means which must be adapted to the individual case. It is extremely

difficult to provide these measures for the patients generally. For example, whether we believe in electricity or not, it is one of the generally recognized means. The galvanic current is the form to use, yet we find that there are very few people who can go to a patient's house and give the galvanic current efficiently. Additional facilities are required and the question of braces, and perhaps later operations, etc., must be considered. Let me urge that these cases be watched over by some one skilled in orthopedics and possessing an infinite degree of perseverance and patience. My experience is that improvement does not cease at the end of two years—said to be the convalescent period—but that they will continue to improve as long as we continue to work with them throughout a number of years.

Apparatus is used for the prevention of deformity and to facilitate locomotion. Even when used constantly it does not hinder in any way the recovery of the child. I know of no cases in which so much can be done as for patients such as these in which the paralysis is often very marked.

Dr. Charles K. Mills: I have listened with great interest to the papers presented here this evening; certainly, individually and as a society we should be grateful for the data presented. Recently I presented my views regarding this disease, and especially with reference to its epidemiology and probable etiology, before another society in this city, and I do not feel tonight that I am required to take other points of view than those then maintained. Especially referring to the question of the communicability of the disease, my views have already been largely expressed in the remarks of Dr. Weisenburg. I do not believe it is a disease which we should term "seriously contagious," which expression perhaps is not as accurate as it should be. It is an infectious disease, and probably somewhat or slightly transmissible by human contact.

The first great propagandist of the doctrine of the personal contact theory of communicability was Wickman. His book, while admirable, is defective in some respects. It is striking that in it, notwithstanding its great merits and that he protests against the doctrine of insectile transmission and favors the personal contact theory, he gives scarcely a fact in connection with the environmental condition of the localities in which his hundreds of cases occurred. Wickman, like others, spends much space in his chapter on epidemiology in discussing the fact that the disease spreads along lines of travel. This argument is open to very evident objections. He gives an account of school-houses with schemes and diagrams of the manner in which the disease radiated from these schools regarded as foci, but he fails to give us any facts, so far as I recall his observation, with regard not only to the condition of these schools and

their environment, but also the condition of the houses and the localities from whence came the children who went to these schools.

During this epidemic, with an entomologist and others, I made many personal investigations of the localities in which the cases occurred. This investigation included between 300 and 400 cases occurring in about twenty wards of the city and in Chester, Montgomery, and Delaware Counties. In 90 per cent. of the localities studied we were struck by the presence of insanitation and insectile life. The investigation was carried on especially by Mr. Bodine regarding the character and number of insects and animals.

I am glad to hear from Dr. Lavinder that the United States Public Health Service has been doing field work upon this subject, and I look forward with great anticipation to not only the publication of this field work, but to that which I have no doubt we shall have from New York, Brooklyn and elsewhere. Up to the present time far too little has been done in field work in the study of this disease. No man holds in higher respect than I do good laboratory and good clinical work. In connection with the thirty-five to forty diseases, human and of lower animals, however, determined to have been caused by insects, in the vast majority the host, the carrier, and the nature of the disease have been largely revealed by good field work in the localities where the disease occurred.

Dr. Weisenburg has spoken of the question of multiple cases, a subject which interested me very much in the Philadelphia epidemic. These multiple cases occur in a way which indicates that they are due to a common infection in the majority of instances. Some of the writers favoring the contact theory have made shorter and shorter the period of incubation in order to carry out their view of personal contact being the only method of transmission.

Dr. H. A. Hare: During the past summer a strict quarantine was established at Girard College. No friends or relatives were allowed to enter the grounds, and no boy was allowed to leave the grounds. Only one boy came down with poliomyelitis—and very promptly died of it—during the entire period of the last epidemic. The janitor of the building in which this boy and about forty-nine other boys lived asked permission of the woman in charge of the building to go home because his child was ill. Her suspicions were immediately aroused and she told him that as long as the child remained sick not to return to work. Investigation showed that the child had been taken to the Municipal Hospital that afternoon suffering from acute poliomyelitis. The supposition therefore was that this janitor, coming from the sick child at home, had brought the infection to this boy in Girard College and that this was the explanation

of the spread of the disease. This theory, however, was negatived by reason of the fact that several of the boys in the College hearing about this child being desperately ill, came to the infirmary worried about themselves because they had used a mouth organ belonging to the sick boy the day he was taken ill, so that they had both blown out and inhaled the infection. It would seem, therefore, that these boys were immune and that the janitor probably did not bring in the infection.

It is my opinion that we shall never have control of these epidemics until we have Federal control. I say this because of my observation last summer. Orders were given in Rhode Island to quarantine against "suspected districts," meaning New York. At the time quarantine was established there was a larger proportion of cases to the total population in Newport than in New York, but Newport was not quarantined. A health officer of the State warned the hotel-keepers that if such quarantine were instituted, business would be hurt, and appealed to them to do nothing to keep people out of Rhode Island. If the State of Rhode Island had been under Federal control, local people would have had no political pull to interfere with proper ordinances. The thing reached the height of absurdity when a certain number of inspectors were ordered to board and walk through trains and determine whether any of the children on board had poliomyelitis.

Regarding the outbreak of these local epidemics, three years ago in Jamestown there occurred only 4 cases of poliomyelitis, and 3 of these cases lived five miles away from the fourth. These were apparently isolated epidemics. There was only one case of a child being taken ill in the town of Jamestown. The others were near the lighthouse at Beaver Tail.

Regarding the transmissibility of the disease, the question is whether some of our difficulties do not lie in the fact that the disease is transmissible only at certain periods of its existence. At one time we did not know that yellow fever was transmissible until after a certain stage of development. I have been wondering whether, for example, in the case of this child at Girard College, infection may not have been brought to it at a certain stage of development; whereas, the boys who used the mouth organ got the microörganisms at a period when the infection was not of a transmissible nature.

Dr. A. C. Abbott: With all due respect to the opinions that have been expressed upon the mode of transmission of this disease, I feel that the matter is still unsettled. There is one question which does not seem to have been satisfactorily answered, and that is with regard to its seasonal occurrence. The chart which Dr. Emerson has shown is the one which we know as the ordinary chart of poliomyelitis, occurring with its maximum

about midsummer and falling off toward the early autumn, and disappearing as cold weather comes on. We are also aware that there are occasional exceptions to this, the one occurring in Sweden in the winter being a conspicuous illustration. I know of no natural law to which there is not an occasional exception, nor do I recall the circumstances surrounding the outbreak in Sweden in cold weather. In the cases occurring here in Philadelphia there is little which points directly to transference of the disease from one individual in a family to another, or to the human being as an agent for the carrying of the disease.

I am not sure what value it has, but I think it is significant and well worth noting that during the course of the epidemic here in Philadelphia an investigation by the Health Department brought out the fact that a large majority of the cases were in those parts of the city having the largest number of stables, and were often located close to stables. That may be only a coincidence, but still I think it is worth bearing in mind. Another interesting thing in connection with it was that, as the epidemic began to fall off, the demand arose for the opening of our schools, and the epidemic had by no means entirely disappeared in Philadelphia when the schools were opened. We were having in September, when the schools opened, from 7 to 10 new cases of poliomyelitis a week. With the opening of the schools about 250,000 children of the public and parochial schools were brought together, and I, for one, looked upon it as a rather hazardous experiment. As a matter of fact, nothing happened of a serious nature. There was no increase in the number of cases. They have, rather, steadily diminished, and at the present time we have no more cases of poliomyelitis in Philadelphia than we have had in years that were not epidemic years. Among the 250,000 school children brought together I think it reasonable to suppose there must have been some carriers, some unrecognized cases, and yet there was no spread. Dr. Hare touched upon a point I had in mind. It seems to me that at the present time we occupy about the same mental attitude on the transmissibility of this disease as on that of yellow fever before we know how it arises and is carried. There are men in the room who will remember the various opinions expressed regarding the transmission of yellow fever before we knew the mode of its transmission. There were strong arguments offered in favor of direct transmission, and arguments just as strong in opposition to that view. I do not doubt that direct transmission occurs from person to person, or that we have human carriers, but I do not believe those are the only ways the disease is spread.

Dr. John A. Kolmer stated that since the appearance of anterior poliomyelitis in Philadelphia his assistants, Doctors Freese, Brown, Matsunami, and Meine and he had conducted investigations in the labora-

tory of the Philadelphia Hospital for Contagious Diseases, and lately with financial assistance from a fund under the charge of Dr. Charles K. Mills, along two distinct lines: (1) to detect if possible, by studies on the cerebrospinal fluid and blood, characteristic changes which occur early enough and are sufficiently constant to be of diagnostic value; this is especially important in view of the atypical forms of the disease, the great damage which may result before a clinical diagnosis is possible and because therapeutic measures are more likely to be efficacious if given early, and particularly intraspinal injections of immune serum; (2) to search for the etiological agent of the disease in the blood, cerebrospinal fluid, and tissues by bacteriological methods.

Studies with the cerebrospinal fluid were planned to detect the earliest evidences of inflammatory changes in the meninges. Total and differential cell counts were made of over 700 fluids; a perfectly clear fluid with a slight increase of cells was found to be the most constant change. In their experience the majority of counts ranged from 12 to 100 cells per cubic millimeter. Differential counts showed in most instances an increase of the small lymphocyte variety of cell, but no cell or cells characteristic of this infection alone. Protein tests were conducted with about 900 fluids and after various methods. The method of Nonne yielded but 11 per cent. positive reactions; that of Noguchi 26 per cent., while the method of Gordon and that of Kaplan proved more delicate and yielded 72 and 97 per cent. positive reactions, respectively. Their results substantiate the observations of Dr. Flexner and his associates, that a clear fluid showing an increase of cells and protein is suggestive of poliomyelitis. Determinations of the protein content by reduction tests, as that of Materhofer and the Lange's colloidal gold reaction, yielded interesting results, but were not found to possess diagnostic value. Qualitative tests for dextrose lead them to believe that this substance is reduced in amount in about 30 per cent. of cases.

Immunological studies consisted of complement-fixation experiments with cerebrospinal fluids and blood sera and a number of salt solution antigens of brain, cord, liver, spleen, mesenteric glands, thymus, suprarenal glands, etc.; with both cerebrospinal fluids and sera slightly positive reactions were observed in from 6 to 16 per cent. with antigens of the brain and cord; with antigens of poliomyelitic spleens, positive reactions were observed with 36 per cent. of fluids, but only with about 3 per cent. of sera; control antigens prepared of the spleens of rabbits and persons succumbing to diseases other than poliomyelitis yielded positive reactions with about 8 per cent. of fluids. We have no explanation to offer at the present time for the reactions with splenic antigens.

Tests for natural antisheep hemolysin and complement in the cerebro-

spinal fluid as evidence of meningitis with increased permeability of the meninges were positive in from 30 to 66 per cent. of cases.

Experiments on the total toxicity of the cerebrospinal fluids from cases of poliomyelitis yielded negative results. Our researches have not disclosed any constantly characteristic changes in the cerebrospinal fluids of sera of poliomyelitis; in instances it is impossible to differentiate the fluid of poliomyelitis from early tuberculous meningitis; it is easier to differentiate from the fluid of meningismus.

Their bacteriological studies were conducted after the method described by Dr. Flexner and Dr. Noguchi. Cultures were made of the cerebrum, pons and medulla, cord, and the internal organs, as the liver, spleen, kidneys, thymus gland, suprarenal glands, and enlarged mesenteric glands. Early in their investigations they found that a high percentage of these cultures showed the presence of streptococci and diplococci, and in a few instances diphtheria-like and Gram-negative bacilli. For example, streptococci were found in 50 per cent. of the brains and cords of 8 cases; this organism was also found in all of the organs mentioned, but not in every case. Influenced by the findings of the earlier investigations, among whom may be mentioned those of Geirswold, Flexner and his associates, Dixon, Fox and Rucker and others, and as these microorganisms were regarded as of no etiological consequences, they did not study them further until various reports were made claiming that these cocci produce poliomyelitis among the lower animals. Thirty-five different cultures of these streptococci, 5 of the diplococci, 5 of the diphtheroids, and 9 of the Gram-negative bacilli were injected intravenously and intraperitoneally into rabbits, and most of the cultures of streptococci were also injected intracerebrally, but without producing the least evidences of poliomyelitis in any of these animals. Arthritis among the rabbits injected with streptococci has developed; also a number have succumbed with a pleuritis and pericarditis, but in no instance were there any evidence of paralysis or histological evidences of poliomyelitis in the brain and cords. Streptococci have also been injected intracerebrally, intravenously, and intraperitoneally into monkeys without producing poliomyelitis; one monkey developed a severe streptococcic meningitis from which it is gradually recovering. This animal refused to use its hind legs, which were kept constantly flexed on the abdomen, but we were convinced that true paralysis was not present.

Our streptococci requires at least ten days for appreciable multiplications in ascites broth under anaërobic conditions, but all of them grow profusely in aërobic cultures. Fresh aërobic cultures show a rather large type of streptococcus; the same cultures grown anaërobically over a period of two or three weeks show marked changes, consisting mainly of a reduc-

tion in size and a tendency to lose the power of retaining the Gram stain. Smears of these cultures show chains of large and very small cocci side by side, as described by Rosenau. Large Chamberland filters have not permitted their filtration in our experience; all of these microörganisms have been found viable after the tissues had been preserved in a 50 per cent. solution of glycerin for a period of three to five weeks.

Anaërobic blood cultures of 20 cases of poliomyelitis showed the presence of a streptococcus in 1 case and of staphylococci in 13 cases; anaërobic cultures of the cerebrospinal fluids of 99 cases of poliomyelitis showed streptococci in none and a staphylococcus or diplococcus in 46 cases.

Our investigations have shown therefore that a streptococcus and a diplococcus occur in a large percentage of cases of poliomyelitis, and not only in the brains and cords but also in various internal organs. Our experiments have failed without exception to produce experimental poliomyelitis with these cultures. At the present time we have a monkey injected intracerebrally with 10 c.c. of a sterile filtrate of a poliomyelitic cord containing living streptococci, but sufficient time for the development of poliomyelitis has not yet elapsed. We have taken smears of the organism discovered by Dr. Flexner and Dr. Noguchi and kindly sent by Dr. Flexner and stained with a blue dye and decolorized them thoroughly followed by an intense Gram stain without counterstain. In these smears we find what we believe are streptococci; as described by Dr. Flexner the cocci are very small. As previously stated, we have found small cocci in our old anaërobic cultures, but Dr. Flexner's organism is grown with difficulty and produces poliomyelitis in monkeys, whereas our streptococci grow readily, and in all of our experiments to date have failed to produce poliomyelitis. It appears evident to us, however, that streptococci and a diplococcus are widely distributed in poliomyelitis, and at present we regard them as secondary invaders, growing in symbiosis with the true organism in a manner analogous to the growth of streptococci in persons suffering with scarlet fever.

Dr. A. A. Eshner: Among many interesting and important statements that have been made tonight attention may profitably be directed to that with respect to the occurrence of cases in which paralysis does not develop. Admitting this fact, we have here an explanation in part for the relatively slight susceptibility to the disease, inasmuch as there may be numerous cases of this sort, and we have learned that an attack of the disease confers immunity to subsequent attack. Also we may have here an explanation of the cessation of the disease, through exhaustion of the soil. This fact further may have a possible bearing with respect to the future. The fear naturally has arisen, in view of the occurrence of the epidemic

this year and its subsidence with the advent of cold weather, that we may have a recrudescence in the ensuing year. On the basis of the statements that have been made it may be found that in the ensuing year there will be even a greater degree of immunity to the disease than in the past, and it is therefore to be hoped that we may encounter a smaller number of cases next year than in the present.

Dr. Emerson (closing): We have certain proved data upon this subject, but we are not ready to speak positively upon the subject now. In New York we have on Barren Island the most insanitary conditions, and of the 350 children, there was not a single case of poliomyelitis. I know of no combination of conditions so favorable as those prevailing on Barren Island if physical environment is responsible for the spread of infection. Under our modern control we consider the school as our main point in the control of communicable diseases of children, and not as a site of communication of disease. Schools are points of advantage against contagion. No children were accepted in the public or parochial schools from homes in which the disease occurred, nor would we accept children from homes in which diphtheria or scarlet fever existed unless they had previously had the disease. Our institutions were exposed to exactly the same environment as was the rest of the city. Here there was an occasional case of direct contact. Our most exhaustive study of possible insect carriers showed that we could attach no suspicion to any of the insects. Until we are able to detect the vary large number of non-paralytic cases which undoubtedly exist during an epidemic, we are in no position to say there are no secondary cases in poliomyelitis occurring about as commonly as in other communicable diseases.

Our nurses do not, so far as we can detect, serve as carriers for diphtheria, scarlet fever, or typhoid, nor do they commonly become infected in the course of their work among these diseases. This does not seem that we do not regard these diseases as readily communicable. Hospitals provide an environment inimical to the transmission of the disease. Not a single doctor or nurse was infected with poliomyelitis in our hospitals. We held clinics and maintained open house for physicians all through the summer; also physicians in charge of private hospitals had clinics, so that we all gave opportunity to a great many physicians to study the clinical aspects of the disease. We warned every parent on the discharge of the child from the hospital to put it under the care of someone competent to care for an orthopedic case. Every case discharged is being followed by the Committee on After-care, so that each case will be under the care of a competent orthopedic surgeon, usually in coöperation with a neurologist. The after-care of our cases includes (1) the attendance of a

competent orthopedic surgeon; (2) instruction of the guardian of the child in its care; (3) provision of transportation when required; (4) education so that these children may be self-supporting when they reach their majority.

The organization and supervision of this after-care work has been made possible by the generosity of the Rockefeller Foundation.

Dr. Simon Flexner: Those of us who have come from New York have profited very much by the full discussion and the many sides of the problem presented. It seems to me that in closing the discussion, I might refer exclusively to the question of the mode of transmission. Now it would be presumptuous to contend that we have full and complete knowledge of this subject; but what I want to warn against, is being misled by analogies. Reasoning by analogy is notably unsafe, and I think it unfortunate that the analogy of yellow fever was raised this evening. The solution of the mode of transmission of yellow fever is, of course, one of the greatest contributions to epidemiological research and sanitary science that the centuries have produced. But consider how much more quickly that solution would have been brought about if it had been possible to communicate yellow fever to a lower animal. In that case we should not have had to wait for Dr. Reed and his particular group of self-sacrificing American soldiers and civilians to perform the experiment that was destined to be so illuminating and conclusive. But in the present instance we are dealing with a disease that can be communicated to the monkey in all its forms—abortive, meningitic, and paralytic. For the sake of conviction we may for the moment throw out of consideration the abortive and meningitic types of the disease because they may be in individual instances open to question, but you cannot similarly disregard the paralytic example because it corresponds precisely with what is so commonly seen in human beings, and is everywhere recognized as the type of poliomyelitis, besides which the lesions in the monkey exactly reproduce those present in fatal human cases.

In attempting to explain all the instances of transmission, account must be taken of such circumstances as susceptibility, and even more, ability to trace the source of infection in large and actively moving communities. That all persons are not equally susceptible to most diseases is, of course, a common-place observation; but gradually such tests as that of Schick in diphtheria begin to point the basis of the differences observed. Moreover, in the case of all the communicable diseases several cases will arise at one time, so that within an hour or two, one may find say two or three cases of measles in a given home. But with animals we are in a different situation, and that is the advantage of our position

today in reference to poliomyelitis. Since the infection is communicable the validity of any hypothesis can be tested more or less completely. And while it appears to be true that the stable fly has been made in two notable instances to convey the disease to monkeys, later efforts at verification and repetition of the experiment have all failed. I even doubt whether an isolated instance of the disease, five or ten miles from the nearest recognized cases, is more satisfactorily explained on the basis of the notion of fly transmission.

On the other hand, there is no doubt that the active agent of the disease is thrown off by the human body and in a state suitable for entry in an infective state into other persons of whom a part become infected and ill, and another part merely contaminated and capable of spreading the infective agent more widely. What then is the obvious deduction from this fact? Is it not to do what you can to limit this dissemination by acting on the basis of established knowledge? Should later still fuller and even different knowledge be obtained, then we must act also on that. But it is in the highest degree unsound to pursue a phantom and neglect what is established, and that is what would be the result of striving, in your measures of sanitary control of the disease, at some unestablished even if theoretically conceivable mode of infection, and neglecting to deal with the mode which is established as actually occurring.

A NEW METHOD OF INJECTING THE FACIAL NERVE FOR THE RELIEF OF FACIAL SPASM[1]

By GEORGE M. DORRANCE, M.D.

Because of the rather confusing definitions found in the text-books of medicine, particularly those of a few years ago, we have taken the liberty of giving a brief differential diagnosis between facial spasm and tic, or so-called habit spasms, before going on with our treatment.

A second purpose in doing this is that the treatment of these two affections is entirely different, and while alcoholic injections are ideal in cases of facial spasm, they are entirely contra-indicated in tics.

True facial spasm is a reflex act occurring in a single muscle or a group of muscles, and is thought to be due to some form of irritation in the muscles or the nerves of the spinal or bulbar reflex arc.

Tics, on the other hand, are voluntary contractions which have become a habit. It is a psychoneurosis, and is not a spasm at all. Briassard believes that the synergic and coördinated muscular contractions of tic imply a cortical origin.

In facial spasm the spasm is confined to the mechanism of the motor innervation of the face, and is devoid of voluntary or involuntary control, whereas in tics the movements are always more or less under control of the will and subject to involuntary control under violent excitement or emotion.

The spasm of the true facial type is never a natural movement, and cannot be imitated either by the patient or a second person.

[1] Read March 1, 1916.

In tics the movement, however exaggerated, is a natural one, and can be imitated. As Patrick once said, "In spasm the movement is an anatomical disorder; in tics a physiological one."

At the onset of facial spasm the spasm may be confined to only a part of a muscle. Later it takes the entire distribution of the facial nerve, and no more, and is practically always unilateral.

Tics never involve only a part of a muscle, and rarely involve all the facial muscles at any given time, but take associated groups of muscles, varying these from time to time. It is often bilateral.

Facial spasms are painful and incapacitate their victims, whereas tics give a certain pleasure or sense of relief and never interfere with any of the bodily functions, *e. g.*, talking, etc.

Spasms are found in the mentally strong; tics in the neuropathic type of individuals in whom other mental stigmata exist.

There is another form of tic occurring after facial palsy where the tic is synchronous with the winking. This form, likewise, should never be injected.

Alcohol Injection. Our reasons for devising a new method for injecting the facial nerve are two:

1. The older methods are inaccurate (often requiring as many as ten punctures to hit the nerve).

2. The methods are dangerous, *i. e.*, the jugular vein is very apt to be hit.

We formed these conclusions after careful clinical observations, and performed 300 injections of the facial nerve at the stylomastoid foramen on cadavers.

Through the courtesy of Dr. Greeman we made a series of measurements on fifty skulls at the Wistar Institute, and present the following measurements:

On fifty skulls the distance between the stylomastoid foramen and that of the jugular varied 0.2 cm. to 1.2 cm., the average distance being 0.6 cm.

One can readily see how in the older method of simply holding the needle in horizontal position and inserting the point into the stylomastoid foramen that less than 0.5 cm. separates the point of the needle from the jugular vein.

FIG. 1.—Showing needle in proper position.

FIG. 2.—Showing needle in position on skull.

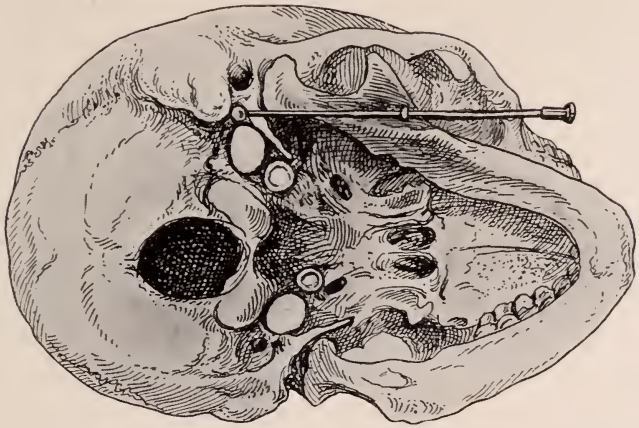


FIG. 3.—Note how the styloid process prevents the needle from going too far inward, thus preventing any possibility of hitting the jugular vein.

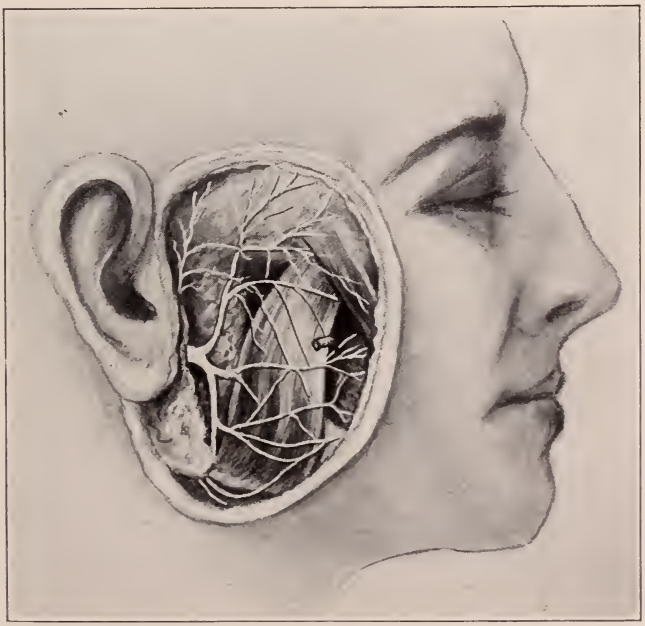


FIG. 4.—Distribution of facial nerve. Note the difficulties offered to the injection of the branches.

Fig. 1 shows the needle in position in old method. Note how close the point is from the jugular foramen.

To recapitulate, because of these difficulties and dangers we devised a method whereby it would be impossible to hit the jugular vein and yet permit one to accurately inject the nerve at its foramen of exit from the skull.

The method is as follows: The needle (trocar and stylet style) is 10 cm. long and 0.4 cm. thick, graded in centimeters.

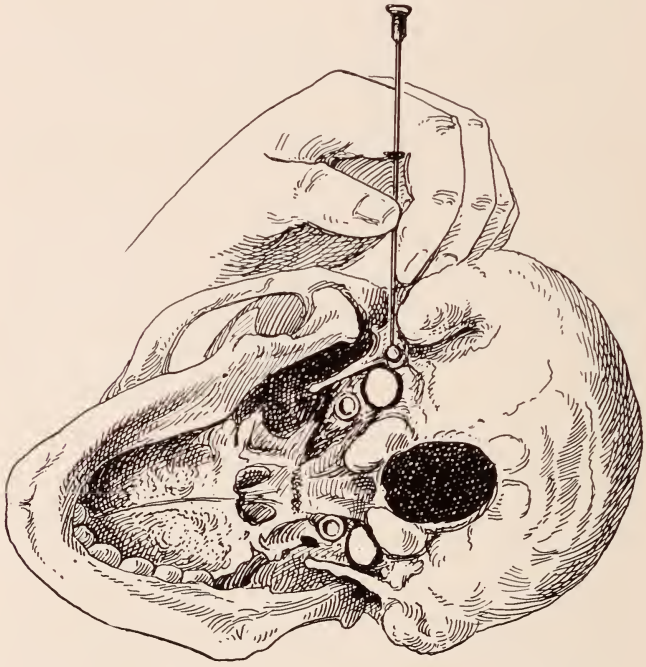


Fig. 5

This needle is inserted at the angle of the jaw (the skin having previously been painted with 5 per cent. tincture of iodin and anesthetized with 0.5 c.c. novocain) and directed backward and upward until the point impinges on the base of the mastoid. This can be made certain of by feeling the point under the skin by the index finger, which is held over the mastoid.

The handle of the needle is elevated and the point is depressed, feeling carefully your way down, and at the same time pushing the point backward and inward until the needle feels its way into the stylomastoid foramen.

The stylet is now removed. If any blood flows out, do not inject, as the point may have pierced a small vein. Withdraw, clean the needle, and puncture again. If no bleeding occurs, inject a few drops of alcohol. If you are successful in hitting the nerve, immediate facial paralysis occurs. Now inject about 2 c.c. more of alcohol.

Remove the needle. No dressing is needed.

As a rule the patient complains of some pain, which is relieved by hot compresses.

The eye should be protected by goggles and washed with boric acid solution at least twice a day.

For some little time after the injection goggles should be worn when the patient is outside of the house.

It differs from the fifth nerve injections in that we have no trophic changes or corneal ulcers. We must, however, remember that because of the paralysis winking does not occur, hence the cornea is apt to become dry, due to lack of proper irrigation from tears.

Moving the eye from side to side and gentle massage will help to prevent any deleterious results. The face should be massaged daily to prevent atrophy of the muscles.

The method of injection appears to us to be extremely simple, and one or two hours' practice in the dissecting room to train the fingers in the sense of touch, a feature so essential in all injection methods, will render any physician proficient.

We have found from our records that the average depth of the needle was 5 cm., but the distance varies according to the point of insertion and the amount of adipose tissue present on the face of the individual injected.

By this technic the entire distribution of the seventh nerve is paralyzed.

We have on several occasions injected one of the branches of the

seventh going to one or more muscles by injecting the branches anterior to the parotid gland.

It is much more difficult to inject the branches, as the exact plane of the nerve must be hit, as alcohol does not diffuse through facial planes.

The results from the injection of the branches do not compare as favorably as those obtained by injection at the stylomastoid foramen. It appears as if it were necessary to rest the entire distribution of the seventh to effect a perfect result.

Results and Conclusions of Alcoholic Injections. 1. Most patients will willingly exchange a spasm for a paralysis.

2. The paralysis is only temporary. Voluntary motion slowly returns until complete function is restored.

3. The spasm never recurs at the time of the return of voluntary power, but varies from a month to several years. In some cases it never recurs.

4. In the event of spasm recurring, patients are willing to have injection repeated. (Return of power slower with each subsequent injection.)

5. For injection at the stylomastoid foramen we use 2 c.c. of 70 per cent. alcohol. For injection of the branches not over 50 per cent. alcohol, because of the danger of sloughing, which is prone to occur in loose fatty tissue when injected superficially.

DISCUSSION

Dr. Alfred Gordon: On November 12, 1914, I presented before the College a paper with a very similar title, and presented pictures of faces before and after treatment. Dr. Dorrance has covered the entire ground. There are only two points which I might perhaps emphasize. First, that we need have no fear whatever in injecting alcohol into a motor nerve. Several years ago I made some experiments in dogs and the work was published with illustrations in the *Journal of Nervous and Mental Diseases*. The work was undertaken to determine whether alcohol was capable of producing damage when injected into a nerve. The dogs were

selected and classified. In some the injections were made in the infra-orbital nerve, in others in the sciatic, in others in the facial nerve. The nerves were removed after a certain number of days and examined histologically, not only the nerves but the ganglia with which they were connected. For the facial nerve its nucleus was traced in the medulla. My conclusion was that when a motor nerve is treated with alcohol injected into the sheath or nerve itself no damage is done to the nerve fibers. I found that there was a complete restoration of the paralysis when the facial nerve was injected with alcohol. On the other hand, when the infra-orbital was injected, I found marked trophic disturbance in the area of its distribution. The same was observed with the sciatic nerve. Trophic disturbances in the area of distribution of nerves posessing mixed sensory and motor fibers has also been demonstrated by Briss and Schlösser. No damage is done in purely motor nerves according to my experiments.

The second feature I would emphasize is the point of injection. I am not a surgeon and did not do much exact work upon cadavers as Dr. Dorrance has done, but in some patients I succeeded in injecting at once into the facial nerve. In a number of instances I have had to make several attempts. One practical point gained from my experience is that when I presumably have gone into the facial nerve, if the patient does not flinch, I know I am not in the nerve. On the other hand, the moment the facial nerve is touched there is the flinching of the patient. Another point which Dr. Dorrance, I believe, did not mention is that if you strike the facial nerve you must get absolutely immediate paralysis.

Dr. Dorrance (closing): I did mention in the paper that immediately upon striking the facial nerve the patient winces. Dr. Gordon is right. I became interested in this work because I had failed four or five times in injecting the facial nerve. After going over the subject I found that others had failed also. We are now able to go directly into the nerve. There is nothing new in the paper except the method of striking the nerve. The whole subject is very complicated and involves a discussion of the effect of alcohol on the nerves. My own specimens of nerves into which alcohol was injected are confusing and I do not feel inclined, therefore, to discuss the subject at the present time.

A STUDY OF THE SIGNS AND SYMPTOMS OF AUTONOMIC DISTURBANCE OCCURRING IN PULMONARY TUBERCULOSIS[1]

By MYER SOLIS-COHEN, M.D.

The various manifestations of autonomic disturbance occurring in cases of pulmonary tuberculosis have received little attention, although they are present, to a greater or less degree, in nearly every patient. Indeed, they frequently are more prominent than the symptoms referable to the lungs and, especially in latent and arrested cases, may be the only phenomenon noticed by the patient or by his physician. On the other hand, unless inquired for, they may be very easily overlooked.

For a number of years I have noted evidences of autonomic disturbance in many patients suffering from pulmonary tuberculosis, and, conversely, have frequently discovered tuberculous lesions in the lungs of patients who had presented phenomena of autonomic ataxia.

Several years ago, while mentioning this coincidence to Dr. Solomon Solis-Cohen, who confirmed its occurrence, I was asked by him to make an investigation in regard to it, but the scheme suggested was so elaborate that I never had the time to undertake it myself; nor could I sufficiently interest and enthuse the resident physicians at the sanatoria I attend to get them to carry it out.

For several years, however, in examining patients with pulmonary tuberculosis, when time permitted, I have been directing

[1] Read February 2, 1916.

my attention to a number of the principal signs and symptoms of autonomic disturbance, noting their presence or absence. This paper presents an analysis of such patients and of those of my tuberculous patients in whom I had previously noted evidence of autonomic disturbance. In few of the latter did I inquire for autonomic symptoms, merely recording those that forced themselves upon my attention; nor did I note the absence of such symptoms.

One hundred and nineteen patients form the basis for this study, of which 41 are male and 78 female. Nineteen were ten years old or younger, 18 were between the ages of eleven and twenty, 45 between twenty-one and thirty years, 14 between thirty-one and forty years, 9 between forty-one and fifty years, 3 between fifty-one and sixty years, and 2 between sixty-one and seventy years. The age was not recorded in 9 cases. Ninety-four were in the first or incipient stage of the disease, according to the schema of the National Association for the Study and Prevention of Tuberculosis, 13 were in the second or moderately advanced stage, and 12 were in the third or far-advanced stage. Ninety-four were in Class 1 of Turban's classification, 16 in Class 2, and 9 in Class 3. Forty were what I term latent cases, presenting no symptoms referable to the lungs, 12 were arrested cases, 32 were improving, 24 stationary, and 12 advancing cases. Forty-eight gave a history of tuberculosis in the family, and 21 gave a history of no tuberculosis in the family, the other histories not being sufficiently definite to warrant their inclusion. Eleven gave a definite history of vasomotor disturbances in the family, while in most of the cases no inquiry had been made as to any such condition.

Thirteen of the patients were under my care at the Home for Consumptives at Chestnut Hill, 27 were under my care at the Eagleville Sanatorium for Consumptives, and 79 were patients in my private practice or seen in consultation. The symptoms of autonomic disturbance particularly inquired for were flushing, burning of one side of the face or of one part of the body, subjective sensations of heat and cold, sweating (exclusive of night-

sweats), bleeding, angioneurotic edema, urticaria, asthma, hay fever, and migraine. Note was made, of course, of any other vasomotor symptoms complained of. The signs indicative of vasomotor disturbance looked for on physical examination were pallor, clearness of skin or the reverse, dermographism, the production of a black line by drawing silver across the face (which must be free from powder), exposure of sclera above or below the cornea in repose, and on voluntarily widening the commissure and on fixing the eyes, or an increased exposure of sclera under the last two conditions, involuntary elevation of the eyebrows on voluntarily widening the commissure, and the division of the finger-nail into three striations of reddish distally, whitish in the middle, and bluish proximally. The size of the thyroid gland was also observed, being recorded as palpable or not palpable, or as enlarged slightly, moderately, or markedly.

Unfortunately, every patient was not examined for each sign and symptom. Few children, for instance, were considered reliable enough to be questioned for subjective symptoms. Frequently, time did not permit a full investigation. I often forgot to look for or to record the presence or especially the absence of one or more of the signs and symptoms. The earlier histories contain no systematic investigation and no negative records, but merely note the presence of symptoms complained of by the patient and of signs brought to the attention of the examiner. In calculating the percentage of patients exhibiting a certain phenomenon, therefore, I have included only those patients who are recorded as showing or not showing it, leaving out those whose histories are silent in regard to it. This probably makes the percentages somewhat higher than they actually are; for it is more likely that a sign or symptom is absent when no mention is made of it than that it was overlooked. On the other hand, the symptoms studied are of such a character that a patient coming to a physician for a cough or for nervousness would not be apt to relate them, even when present, and the signs are not those that would be noticed by a physician not on the lookout for them. I have not regarded

seriously any percentage based on less than 5 cases, which I have indicated by parentheses. Inasmuch as in only a very few cases was mention made of the absence of pallor, of involuntary elevation of the eyebrows on voluntarily widening the commissure, or of vasomotor symptoms other than those mentioned, no attempt has been made to calculate their percentages, the number occurring merely being stated.

With this exception the following tables give the number of cases specifically examined for each sign or symptom and the number of cases in which it was observed, with the percentage of patients showing it. The patients are then divided according to sex, age, stage of the disease, amount of lung involvement (Turban's Classification), progress of the disease, a positive or negative family history of tuberculosis, and a positive family history of vasomotor disturbance.

FLUSHING

	No of cases.	Present in	Per cent.
Total	73	36	49
Male	24	7	29
Female	49	29	59
10 years and less	5	5	100
11 to 20 years	11	5	45
21 to 30 "	32	14	44
31 to 40 "	10	5	50
41 to 50 "	2	1	(50)
51 to 60 "	3	0	(0)
61 to 70 "	2	1	(50)
Stage I	55	30	55
" II	9	3	33
" III	9	3	33
Class 1	57	31	54
" 2	11	4	36
" 3	5	1	20
Latent	27	14	52
Arrested	8	3	37
Improving	14	8	57
Stationary	16	6	37
Advancing	8	5	62
Family history of tuberculosis	29	16	55
No family history of tuberculosis	13	4	31
Family history of vasomotor disturbance	10	9	90

BURNING, ESPECIALLY OF THE ONE SIDE OF THE FACE OR ONE PART OF THE BODY

	No of cases.	Present in	Per cent.
Total	61	16	26
Male	22	1	5
Female	39	15	38
10 years and less	1	0	(0)
11 to 20 years	9	2	22
21 to 30 "	30	7	23
31 to 40 "	9	2	22
41 to 50 "	2	1	(50)
51 to 60 "	3	1	(33)
61 to 70 "	2	0	(0)
Stage I	46	13	28
" II	8	2	25
" III	7	1	14
Class 1	46	13	28
" 2	11	3	27
" 3	4	0	(0)
Latent	25	6	24
Arrested	7	2	29
Improving	9	2	22
Stationary	13	4	31
Advancing	7	2	29
Family history of tuberculosis	24	7	29
No family history of tuberculosis	21	12	57
Family history of vasomotor disturbance	9	4	44

SUBJECTIVE SENSATIONS OF HEAT

	No. of cases.	Present in	Per cent.
Total	59	14	24
Male	21	1	5
Female	38	13	34
10 years and under	1	0	(0)
11 to 20 years	10	1	10
21 to 30 "	23	6	26
31 to 40 "	10	3	30
41 to 50 "	3	0	(0)
51 to 60 "	3	0	(0)
61 to 70 "	2	0	(0)
Stage I	44	14	32
" II	7	0	0
" III	7	0	0
Class 1	45	13	29
" 2	9	1	11
" 3	4	0	(0)
Latent	23	9	35
Arrested	6	2	33
Improving	8	0	0
Stationary	14	3	21
Advancing	7	0	0
Family history of tuberculosis	23	5	22
No family history of tuberculosis	10	1	10
Family history of vasomotor disturbance	9	2	22

Subjective Sensations of Cold

	No. of cases.	Present in	Per cent.
Total	56	15	27
Male	21	3	14
Female	35	12	34
10 years and under	1	0	(0)
11 to 20 years	10	0	0
21 to 30 "	29	10	34
31 to 40 "	7	2	29
41 to 50 "	2	0	(0)
51 to 60 "	3	0	(0)
61 to 70 "	2	1	(50)
Stage I	41	11	27
" II	8	3	37
" III	7	1	14
Class 1	41	12	29
" 2	10	2	20
" 3	5	1	20
Latent	20	7	35
Arrested	5	2	40
Improving	9	0	0
Stationary	15	3	20
Advancing	7	3	43
Family history of tuberculosis	24	8	33
No family history of tuberculosis	10	1	10
Family history of vasomotor disturbance	7	3	43

Sweating (Exclusive of Night-sweats)

	No. of cases.	Present in	Per cent.
Total	65	29	45
Male	24	7	29
Female	41	22	54
10 years and under	1	0	(0)
11 to 20 years	8	1	12
21 to 30 "	33	15	45
31 to 40 "	8	4	50
41 to 50	3	3	(100)
51 to 60 "	2	1	(50)
61 to 70 "	2	0	(0)
Stage I	50	24	48
" II	8	2	25
" III	7	3	43
Class 1	51	25	49
" 2	10	4	40
" 3	4	0	(0)
Latent	29	19	66
Arrested	7	2	29
Improving	6	0	0
Stationary	15	5	33
Advancing	8	3	37
Family history of tuberculosis	26	14	54
No family history of tuberculosis	12	4	33
Family history of vasomotor disturbance	10	8	80

Bleeding

	No. of cases.	Present in	Per cent.
Total	63	16	25
Male	24	4	17
Female	39	12	30
10 years and under	1	0	(0)
11 to 20 years	11	2	18
21 to 30 "	31	11	35
31 to 40 "	8	1	12
41 to 50 "	3	1	(33)
51 to 60 "	3	0	(0)
61 to 70 "	2	0	(0)
Stage I	46	12	26
" II	11	4	36
" III	6	0	0
Class 1	46	12	26
" 2	12	4	33
" 3	5	0	0
Latent	24	8	33
Arrested	9	2	22
Improving	9	2	22
Stationary	15	4	27
Advancing	6	0	0
Family history of tuberculosis	26	9	35
No family history of tuberculosis	11	2	18
Family history of vasomotor disturbance	9	4	44

Angioneurotic Edema

	No. of cases.	Present in	Per cent.
Total	63	13	21
Male	23	2	9
Female	40	11	27
10 years and under	1	0	(0)
11 to 20 years	11	2	18
21 to 30 "	28	8	28
31 to 40 "	10	3	30
41 to 50 "	3	0	(0)
51 to 60 "	3	0	(0)
61 to 70 "	2	0	(0)
Stage I	46	12	26
" II	9	1	11
" III	8	0	0
Class 1	45	11	24
" 2	13	2	15
" 3	5	0	0
Latent	24	7	29
Arrested	8	3	37
Improving	9	0	0
Stationary	14	3	21
Advancing	8	0	0
Family history of tuberculosis	23	7	30
No family history of tuberculosis	12	3	25
Family history of vasomotor disturbance	8	5	62

Urticaria

	No. of cases.	Present in	Per cent.
Total	63	24	38
Male	21	5	24
Female	42	19	45
10 years and under	1	0	(0)
11 to 20 years	10	2	20
21 to 30 "	28	8	29
31 to 40 "	10	8	80
41 to 50 "	3	2	(67)
51 to 60 "	3	1	(33)
61 to 70 "	2	0	(0)
Stage I	46	21	46
" II	9	1	11
" III	8	2	25
Class 1	46	21	46
" 2	13	2	15
" 3	4	1	(25)
Latent	22	12	55
Arrested	8	4	50
Improving	10	2	20
Stationary	16	6	37
Advancing	7	0	0
Family history of tuberculosis	21	10	48
No family history of tuberculosis	12	5	42
Family history of vasomotor disturbance	7	3	43

Asthma

	No. of cases.	Present in	Per cent.
Total	61	9	15
Male	22	3	18
Female	39	6	15
10 years and less	1	0	(0)
11 to 20 years	10	0	0
21 to 30 "	30	3	10
31 to 40 "	10	3	30
41 to 50 "	2	1	(50)
51 to 60 "	3	1	(33)
61 to 70 "	2	0	(0)
Stage I	46	6	13
" II	8	2	25
" III	7	1	14
Class 1	46	6	13
" 2	10	3	30
" 3	5	0	0
Latent	23	4	17
Arrested	6	1	17
Improving	10	1	10
Stationary	13	3	23
Advancing	7	0	0
Family history of tuberculosis	24	3	12
No family history of tuberculosis	13	3	23
Family history of vasomotor disturbance	6	2	33

Hay Fever

	No. of cases.	Present in	Per cent.
Total	59	4	7
Male	22	2	9
Female	37	2	5
10 years and less	1	0	(0)
11 to 20 years	10	0	0
21 to 30 "	29	2	7
31 to 40 "	9	1	11
41 to 50 "	2	1	(50)
51 to 60 "	3	0	(0)
61 to 70 "	2	0	(0)
Stage I	45	2	4
" II	8	1	12
" III	6	1	17
Class 1	44	2	5
" 2	11	2	22
" 3	4	0	(0)
Latent	24	2	8
Arrested	7	0	0
Improving	9	0	0
Stationary	13	2	15
Advancing	6	0	0
Family history of tuberculosis	25	2	8
No family history of tuberculosis	12	1	8
Family history of vasomotor disturbance	6	0	0

Migraine

	No. of cases.	Present in	Present in.
Total	74	32	43
Male	23	2	9
Female	51	30	59
10 years and less	1	0	(0)
11 to 20 years	11	4	36
21 to 30 "	32	13	41
31 to 40 "	11	6	55
41 to 50 "	7	5	71
51 to 60 "	3	0	(0)
61 to 70 "	2	0	(0)
Stage I	57	28	49
" II	9	3	33
" III	8	1	12
Class 1	57	28	49
" 2	12	4	33
" 3	5	0	0
Latent	32	20	62
Arrested	9	3	33
Improving	9	0	0
Stationary	16	8	50
Advancing	7	0	0
Family history of tuberculosis	26	12	46
No family history of tuberculosis	18	10	56
Family history of vasomotor disturbance	9	7	73

Clear Skin

	No. of cases.	Present in	Per cent.
Total	75	64	85
Male	22	17	77
Female	53	47	89
10 years and under	16	13	81
11 to 20 years	14	12	86
21 to 30 "	23	20	87
31 to 40 "	9	9	100
41 to 50 "	3	3	(100)
51 to 60 "	2	2	(100)
61 to 70 "	2	0	(0)
Stage I	64	55	86
" II	6	5	83
" III	5	4	80
Class 1	66	56	85
" 2	6	5	83
" 3	3	3	(100)
Latent	24	21	87
Arrested	10	10	100
Improving	26	22	85
Stationary	10	9	90
Advancing	5	2	40
Family history of tuberculosis	32	29	91
No family history of tuberculosis	8	8	100
Family history of vasomotor disturbance	5	4	80

Demographism

	No. of cases.	Present in	Per cent.
Total	94	82	87
Male	31	29	94
Female	63	53	84
10 years and under	16	15	94
11 to 20 years	16	15	94
21 to 30 "	35	27	77
31 to 40 "	12	12	100
41 to 50 "	5	4	80
51 to 60 "	2	2	(100)
61 to 70 "	2	2	(100)
Stage I	77	66	86
" II	10	9	90
" III	7	7	100
Class 1	79	68	86
" 2	11	10	91
" 3	4	4	(100)
Latent	33	27	82
Arrested	8	7	87
Improving	26	26	100
Stationary	19	14	74
Advancing	8	8	100
Family history of tuberculosis	35	30	86
No family history of tuberculosis	16	13	81
Family history of vasomotor disturbance	9	7	77

Black Line with Silver

	No. of cases.	Present in	Per cent.
Total	73	29	40
Male	26	7	27
Female	47	22	47
10 years and under	16	6	37
11 to 20 years	14	5	36
21 to 30 "	24	10	42
31 to 40 "	8	3	37
41 to 50 "	4	2	(50)
51 to 60 "	1	0	(0)
61 to 70 "	4	1	(25)
Stage I	59	26	44
" II	9	3	33
" III	5	0	0
Class 1	60	26	43
" 2	11	3	27
" 3	2	0	(0)
Latent	20	9	45
Arrested	7	4	57
Improving	27	11	40
Stationary	12	3	25
Advancing	7	2	29
Family history of tuberculosis	28	12	43
No family history of tuberculosis	12	4	33
Family history of vasomotor disturbance	6	3	50

Exposure of Sclera Normally above or below the Cornea

	No. of cases.	Present in	Per cent.
Total	87	42	48
Male	33	15	45
Female	54	27	50
10 years and under	16	11	69
11 to 20 years	15	4	27
21 to 30 "	31	15	48
31 to 40 "	9	6	67
41 to 50 "	6	3	50
51 to 60 "	3	0	(0)
61 to 70 "	2	0	(0)
Stage I	66	30	45
" II	10	7	70
" III	11	5	45
Class 1	69	33	48
" 2	10	6	60
" 3	8	3	38
Latent	26	12	46
Arrested	9	4	44
Improving	28	13	46
Stationary	14	8	57
Advancing	10	5	50
Family history of tuberculosis	34	17	50
No family history of tuberculosis	15	9	60
Family history of vasomotor disturbance	8	4	50

EXPOSURE OF SCLERA OR OF INCREASED AMOUNT OF SCLERA ABOVE OR BELOW THE CORNEA ON VOLUNTARILY WIDENING THE COMMISSURE

	No. of cases.	Present in	Per cent.
Total	70	35	50
Male	29	19	66
Female	41	16	39
10 years and under	10	2	20
11 to 20 years	13	6	46
21 to 30 "	29	12	41
31 to 40 "	8	6	75
41 to 50 "	4	3	(75)
51 to 60 "	2	2	(100)
61 to 70 "	1	1	(100)
Stage I	54	25	46
" II	8	4	50
" III	8	6	75
Class 1	55	24	44
" 2	8	6	75
" 3	7	5	71
Latent	23	11	48
Arrested	8	5	62
Improving	20	7	35
Stationary	12	8	67
Advancing	7	4	57
Family history of tuberculosis	27	12	44
No family history of tuberculosis	13	9	69
Family history of vasomotor disturbance	6	2	33

EXPOSURE OF SCLERA OR OF INCREASED AMOUNT OF SCLERA ABOVE OR BELOW THE CORNEA ON FIXING THE EYES

	No. of cases.	Present in	Per cent.
Total	73	10	14
Male	31	7	23
Female	42	3	7
10 years and under	13	2	15
11 to 20 years	12	0	0
21 to 30 "	30	4	13
31 to 40 "	6	0	0
41 to 50 "	4	3	(75)
51 to 60 "	2	0	(0)
61 to 70 "	1	1	(100)
Stage I	55	4	7
" II	9	4	44
" III	9	2	22
Class 1	57	4	7
" 2	8	3	37
" 3	8	3	37
Latent	25	3	12
Arrested	7	0	0
Improving	22	2	9
Stationary	12	3	25
Advancing	7	2	29
Family history of tuberculosis	29	6	21
No family history of tuberculosis	12	2	17
Family history of vasomotor disturbance	6	0	0

TRICOLORED NAILS

	No. of cases.	Present in	Per cent.
Total	97	62	64
Male	35	21	60
Female	62	41	66
10 years and under	55	11	20
11 to 20 years	16	11	69
21 to 30 "	39	23	59
31 to 40 "	11	7	64
41 to 50 "	6	5	83
51 to 60 "	3	1	(33)
61 to 70 "	2	2	(100)
Stage I	76	49	64
" II	12	6	50
" III	9	7	73
Class 1	76	50	66
" 2	14	6	43
" 3	7	6	67
Latent	33	22	67
Arrested	9	2	22
Improving	27	22	81
Stationary	19	9	47
Advancing	9	7	73
Family history of tuberculosis	38	22	58
No family history of tuberculosis	17	13	76
Family history of vasomotor disturbance	9	6	67

THYROID GLAND

	No. of cases.	Not palpable.	Per cent.	Palpable.	Per cent.	Slightly enlarged.	Per cent.	Moderately enlarged.	Per cent.	Markedly enlarged.	Per cent.
Total	99	24	24	36	36	17	17	17	17	5	5
Male	35	8	23	15	43	10	29	2	6	0	0
Female	64	16	25	21	33	7	18	15	39	5	13
10 years and under	19	4	21	11	58	3	16	1	5	0	0
11 to 20 years	16	2	12	3	19	5	31	5	31	1	6
21 to 30 "	38	9	24	12	32	6	16	7	18	4	11
31 to 40 "	10	4	40	4	40	0	0	2	20	0	0
41 to 50 "	7	1	14	5	71	1	14	0	0	0	0
51 to 60 "	2	2	(100)	0	(0)	0	(0)	0	(0)	(0)	(0)
61 to 70 "	2	2	(100)	0	(0)	0	(0)	0	(0)	0	(0)
Stage I	79	18	23	26	33	14	18	16	20	5	6
" II	12	4	33	5	42	2	17	1	8	0	0
" III	8	2	25	5	62	1	13	0	0	0	0
Class 1	78	17	22	27	36	14	18	15	19	5	6
" 2	15	6	40	5	33	2	13	2	13	0	0
" 3	6	1	17	4	66	1	17	0	0	0	0
Latent	34	8	24	11	32	4	12	8	24	3	9
Arrested	9	1	11	4	44	3	33	0	0	1	11
Improving	27	5	19	10	37	7	26	5	19	0	0
Stationary	20	6	30	7	35	2	10	4	20	1	5
Advancing	9	4	44	4	44	1	11	0	0	0	0
Family history of tuberculosis	37	7	19	16	43	4	11	8	22	2	5
No family history of tuberculosis	16	6	38	3	19	4	25	2	12	1	6
Family history of vasomotor disturbance	8	2	25	3	37	1	12	1	12	1	12

	Pallor.	Involuntary Elevation of Eyebrows on Voluntarily Widening Commissure	Other Vasomotor Symptoms
	Present in	Present in	Present in
Total	36	24	28
Male	7	13	4
Female	29	11	24
10 years and less	2	3	0
11 to 20 years	6	4	2
21 to 30 "	21	9	13
31 to 40 "	5	4	7
41 to 50 "	1	2	2
51 to 60 "	1	1	0
61 to 70 "	0	0	0
Stage I	31	18	25
" II	2	4	2
" III	3	2	1
Class 1	32	19	26
" 2	2	3	2
" 3	2	2	0
Latent	19	7	15
Arrested	6	5	1
Improving	3	8	1
Stationary	4	3	10
Advancing	4	1	1
Family history of tuberculosis	14	12	9
No family history of tuberculosis	7	4	7
Family history of vasomotor disturbance	3	1	7

In the order of their frequency the autonomic phenomena most commonly observed in patients with pulmonary tuberculosis are dermographism (87 per cent.), clear skin (85 per cent.), tricolored nails (64 per cent.), exposure of sclera or of increased amount of sclera above or below the cornea on voluntarily widening the commissure (50 per cent.), flushing (49 per cent.), exposure of sclera normally above or below the cornea (48 per cent.), sweating, exclusive of night-sweats (45 per cent.), migraine (43 per cent.), black line with silver (40 per cent.), urticaria (38 per cent.), palpable thyroid gland (36 per cent.), subjective sensations of cold (27 per cent.), burning, especially of one side of the face or one part of the body (26 per cent.), bleeding (25 per cent.), subjective sensations of heat (24 per cent.), non-palpable thyroid gland (24 per cent.), angioneurotic edema (21 per cent.), slight enlargement of thyroid gland (17 per cent.), moderate enlargement of

thyroid gland (17 per cent.), asthma (15 per cent.), exposure of sclera or of increased amount of sclera above or below the cornea on fixing the eyes (14 per cent.), hay fever (7 per cent.), and markedly enlarged thyroid gland (5 per cent.).

Many of the phenomena seemed influenced by sex. The percentages in males and females did not differ over ten points in asthma, hay fever, dermographism, exposure of sclera normally, tricolored nails, non-palpable thyroid gland, and palpable thyroid gland. The marked differences, beginning with the greatest, are migraine: males 9 per cent., females 59 per cent.; burning: males 5 per cent., females 38 per cent.; moderately enlarged thyroid gland: males 6 per cent., females 39 per cent.; flushing: males 29 per cent., females 59 per cent.; subjective sensations of heat: males 5 per cent., females 34 per cent.; exposure or increased exposure of sclera on widening the commissure: males 66 per cent., females 39 per cent.; sweating (exclusive of night-sweats): males 29 per cent.; females 54 per cent.; urticaria: males 24 per cent., females 45 per cent.; subjective sensations of cold: males 14 per cent., females 34 per cent.; black line with silver: males 27 per cent., females 47 per cent.; angioneurotic edema: males 9 per cent., females 27 per cent.; exposure or increased exposure of sclera on fixation: males 23 per cent., females 7 per cent.; bleeding: males 17 per cent., females 30 per cent.; markedly enlarged thyroid gland: males none, females 13 per cent.; clear skin: males 77 per cent., females 89 per cent.; slightly enlarged thyroid gland: males 29 per cent., females 18 per cent. Of those not calculated in percentages, pallor was noted in 7 males and 29 females, other vasomotor symptoms in 4 males and 24 females, and involuntary elevation of eyebrows on voluntarily widening the commissure in 13 males and 11 females. Every phenomenon appeared more frequently in females than in males, except the exposure or increased exposure of the sclera on widening the commissure and on fixing the eyes, raising the eyebrows on widening the commissure, and slight enlargement of the thyroid gland.

The division into different age periods also shows some differences. Subjective sensations of cold, sweating, and especially tricolored

nails and exposure or increased exposure of the sclera above and below the cornea on widening the commissure were less frequent in children under eleven years, as were moderate enlargement of the thyroid gland, which was also less between the ages of forty-one and fifty, and marked enlargement of the thyroid, which was also less between the ages of thirty-one and fifty. Flushing appeared more frequently in children under eleven, as did exposure of the sclera above or below the cornea normally, which also appeared more frequently between the ages of thirty-one and forty, and palpable thyroid gland, which also appeared more frequently between the ages of forty-one and fifty. Subjective sensations of heat and hay fever were experienced less often under the age of twenty-one, and exposure of the sclera above or below the cornea normally and a palpable thyroid gland less often between the ages of eleven and twenty. Slight and moderate enlargement of the thyroid gland, however, both occurred more frequently between the ages of eleven and twenty. Inability to palpate the thyroid was observed least between the ages of eleven and twenty and of forty-one and fifty, and bleeding least below twenty years and especially between thirty-one and forty. Urticaria was observed most seldom below thirty years, but especially below twenty years, and dermographism was noted least between twenty-one and thirty years. Bleeding, marked enlargement of the thyroid, pallor, elevation of the eyebrows on widening the commissure, and other vasomotor symptoms were most marked in the age period between twenty-one and thirty, and angioneurotic edema between twenty-one and forty. None of the patients between the ages of thirty-one and forty had an enlarged thyroid, while in that age period asthma, hay fever, clear skin, dermographism, exposure or increased exposure of sclera above or below the cornea on widening the commissure, and a non-palpable thyroid were most frequently found. Urticaria and migraine were most pronounced between thirty-one and fifty, the former especially between thirty-one and forty and the latter especially between forty-one and fifty. Tricolored nails occurred most frequently between the ages of forty-one and fifty years.

The stage of the disease had an influence on a number of the phenomena. Subjective sensations of heat, angioneurotic edema, pallor, raising of the brows on widening the commissure, moderate and marked enlargement of the thyroid gland, and other vasomotor symptoms occurred chiefly in patients in the first stage. Flushing, migraine and a black line with silver, while most common in first-stage cases, also occurred in many patients in the second stage; while urticaria, which also affected mostly patients in the first stage, was observed to a considerable extent in the third stage. Burning affected chiefly first- and second-stage patients, as did bleeding, which was more common in the second stage. Sweating and tricolored nails were phenomena of both the first and second stages; while, all three stages showed a clear skin, slight enlargement of the thyroid gland, and dermographism, the latter occurring most frequently in the third stage and next in the second stage. Most frequent in patients in the second stage were subjective sensations of cold, asthma, normal exposure of the sclera above or below the cornea, and exposure or increased exposure on fixation, and a thyroid gland that could not be palpated. Hay fever was present in both the second and third stages. Third-stage patients exhibited exposure or increased exposure of sclera above or below the cornea on widening the commissure and a palpable thyroid gland much oftener than the others.

When divided according to the amount of pulmonary involvement, patients in class one were those chiefly affected with flushing, subjective sensations of heat and cold, angioneurotic edema, urticaria, migraine, pallor, raising of the eyebrows on widening the commissure, and other vasomotor symptoms. In the first and second classes burning, sweating, moderate enlargement of the thyroid gland, and a black line with silver were observed, the last being more frequently found in the first class. In all three classes there were present a clear skin, a slightly enlarged thyroid gland and dermographism, the last being most marked in the third class and least in the first. Tricolored nails were observed in the first and third classes. Bleeding, asthma, hay fever, exposure of the sclera above or below the cornea normally, and a non-palpable thyroid occurred chiefly in the second class. In the second and third

classes the sclera above or below the cornea was exposed or more exposed on widening the commissure and on fixing the eyes; while in the third class the thyroid was palpable.

The progress of the disease seemed to exert an influence on some of the phenomena. Flushing, burning, a clear skin, dermographism, a black line with silver, exposure of the sclera above or below the cornea normally and on widening the commissure, elevation of the eyebrows on widening the commissure, tricolored nails, and non-palpable, palpable and enlarged thyroid gland were present in each group. Flushing, however, was met with oftener in latent, improving and especially advancing cases. Burning was somewhat less frequent in latent and improving cases, and elevation of the eyebrows somewhat less frequent in arrested, stationary and especially in advancing cases. A clear skin was observed oftenest in arrested cases and least in advancing cases. Dermographism existed in all the improving and advancing cases and in fewest of the stationary cases. A black line with silver was noted most frequently in arrested cases and least in stationary and advancing cases. Exposure of the sclera above or below the cornea was noted most in stationary and especially in advancing cases when normally present, most in arrested, stationary and to some extent in advancing cases and least in improving cases when occurring on widening, and most in stationary and advancing cases and least in improving cases when occurring on fixing the eyes. The nails were tricolored most often in advancing and especially in improving cases and least often in stationary and especially in arrested cases. A thyroid that could not be palpable was recorded to some extent more in stationary and to a considerable extent more in advancing cases and less in improving and least in arrested cases. A palpable thyroid gland was felt more in arrested and advancing cases and slightly less in latent cases. The thyroid gland was slightly enlarged more frequently in improving and especially in arrested cases and less frequently in the other groups. Pallor was noted chiefly in latent cases. Hay fever and other vasomotor symptoms were present chiefly in latent and stationary cases, hay fever occurring oftener in the latter and vasomotor symptoms oftener in the former. Subjective sensations

of heat, angioneurotic edema, migraine, and a markedly enlarged thyroid gland were noted especially in latent, arrested and stationary cases, migraine occurring more frequently in stationary and especially in arrested cases and marked enlargement of the thyroid in latent and especially in arrested cases. Bleeding, urticaria and asthma were experienced in all but advancing cases, urticaria occurring oftener in stationary and especially in latent and arrested cases, asthma oftener in stationary and less in improving cases. Subjective sensations of cold and sweating appeared in all but improving cases, the former being more marked in latent, arrested, and advancing cases, the latter especially in latent cases. Exposure or increased exposure of the sclera above or below the cornea on fixing the eyes was noted in all but arrested cases, being more prominent in stationary and advancing cases. The thyroid gland was moderately enlarged in latent, improving and stationary cases, especially in the first.

Flushing, subjective sensations of heat and especially of cold, sweating, bleeding, dermographism, a black line with silver, exposure of the sclera above or below the cornea on fixation, palpable and moderately enlarged thyroid gland occurred more frequently in patients with a family history of tuberculosis; while burning, asthma, migraine, pallor, exposure of the sclera above or below the cornea normally and on widening the commissure, elevation of the eyebrows on widening the commissure, tricolored nails, a thyroid not palpable or slightly enlarged were observed oftener in patients with no tuberculosis in the family. Angioneurotic edema, urticaria, hay fever, marked enlargement of the thyroid gland, and other vasomotor symptoms showed no change.

The few patients who gave a family history of vasomotor disturbances exhibited angioneurotic edema three times as often as the patients as a whole, and flushing, burning, subjective sensations of cold, sweating, bleeding, asthma, migraine, and marked enlargement of the thyroid gland about twice as often. They showed practically no difference in regard to subjective sensations of heat, clear skin, exposure of sclera above or below the cornea normally, tricolored nails, and non-palpable and palpable thyroid gland. Urticaria and a black line with silver occurred

a little more frequently and dermographism, exposure or increased exposure of the sclera on widening the commissure, and slight and moderate enlargement of the thyroid gland somewhat less frequently than in the patients as a whole. Hay fever and exposure or increased exposure of the sclera above or below the cornea on fixing the eyes occurred in none, and pallor, elevation of the eyebrows on opening the eyes widely, and other vasomotor symptoms in very few. The number giving a history of vasomotor disturbance in the family never exceeded ten in any group and no note was made as to the absence of such history in any case. Consequently the figures must be regarded merely as suggestive.

Discussion. Signs and symptoms of autonomic disturbance occur quite commonly in pulmonary tuberculosis. One hundred and nineteen patients were examined for twenty-six different phenomena, only those who gave a history of the presence or absence of each phenomenon being included in each investigation. In twenty-three instances the number examined seemed sufficiently large to admit of percentage calculations, the number in those groups varying from fifty-six to ninety-nine and more than half being seventy or over. Seventy-five per cent. or more, had dermographism, a clear skin and a thyroid gland that was palpable or more or less enlarged. Between 50 and 75 per cent. showed tricolored nails and exposure or increased exposure of the sclera above or below the cornea on voluntarily widening the commissure. Between 25 and 33 per cent. exhibited flushing, burning, subjective sensations of cold, bleeding, migraine, sweating, urticaria, exposure of the sclera normally above or below the cornea, a black line with silver, and palpable thyroid gland. In between 14 and 25 per cent. were noted subjective sensations of heat, angioneurotic edema, asthma, exposure or increased exposure of the sclera above or below the cornea on fixing the eyes, a non-palpable thyroid gland, slight enlargement of the thyroid, and moderate enlargement of the same gland. In between 5 and 7 per cent. were observed hay fever and marked enlargement of the thyroid gland.

Eight of the phenomena were little influenced by sex. The percentage of females was more than six times that of males

having migraine, burning, subjective sensations of heat, and moderate and marked enlargement of the thyroid gland. The percentage of females was three times that of males exhibiting angioneurotic edema, and from a little less than twice to a little more than twice that of males showing sweating, urticaria, subjective sensations of cold, a black line with silver, and bleeding. Pallor and involuntary raising of the eyebrows on widening the commissure were also observed much more in females than in males. On the other hand, the percentage of males with exposure or increased exposure of sclera above or below the cornea on fixing the eyes was three times that of females, with exposure or increased exposure on widening the commissure nearly twice that of females, and with slightly enlarged thyroid gland considerably more than the percentage of females.

It does not seem possible to generalize concerning the different age periods. Subjective sensations of heat and cold, sweating, bleeding, exposure or increased exposure of the sclera above and below the cornea on widening the commissure, tricolored nails, and moderate and marked enlargement of the thyroid gland are met with in the young. Urticaria, asthma, hay fever, clear skin, dermographia, exposure of the sclera above or below the cornea normally and on widening the commissure, and a non-palpable thyroid gland were most common between the ages of thirty-one and forty years.

The great majority of the autonomic phenomena occurred most frequently in the first stage, although bleeding, subjective sensations of cold, asthma, exposure of the sclera above or below the cornea normally and on fixation, and a non-palpable thyroid gland were most common in the second stage, and dermographism, exposure or increased exposure of the sclera above or below the cornea on widening the commissure, and a palpable thyroid gland were met with oftenest in the third stage.

In the first class of Turban also more phenomena were observed, although bleeding, asthma, hay fever, exposure of the sclera above or below the cornea normally, and a non-palpable thyroid gland occurred more frequently in the second class and dermographism and palpable thyroid in the third class.

It is rather difficult to analyze the cases when divided according to the progress of the disease. Subjective sensations of heat, angioneurotic edema, urticaria, asthma, migraine and marked enlargement of the thyroid gland occurred chiefly in latent, arrested and stationary cases. Hay fever and other vasomotor symptoms were present and bleeding was more pronounced in latent and stationary cases.

No deductions at all can be drawn from the figures obtained after dividing the cases according to the existence or not of a history of tuberculosis in the family.

While the number of cases giving a family history of vasomotor disturbances is too small to admit of making any generalizations, it would appear that such cases exhibit autonomic subjective symptoms in great excess of tuberculous patients taken as a whole but, with the exception of markedly enlarged thyroid gland, present the objective signs of such disturbance in about the same proportion as other cases.

As to what significance is to be attached to these various signs and symptoms of autonomic disturbance that occur so frequently in pulmonary tuberculosis, I do not feel justified at present in venturing a definite opinion. My feeling is that they are due to an excess in the system of one or more of the internal secretions, due to the stimulation of the thyroid gland and other endocrine glands by the tuberculous poison and are evidences of a defense reaction. In latent and arrested cases it is possible that the endocrine glands, which were stimulated to hypersecretion (and possible hypertrophy) by the tuberculous toxin, still continued to secrete in excess of the normal after the disease was checked, this excessive secretion giving rise to the various phenomena of autonomic disturbance. It is possible that in some of the improving and advancing cases the endocrine glands are also sending large amounts of internal secretions into the system, while in others these glands have become more or less exhausted. Stationary cases seem to me to tend to agree more in their autonomic phenomena with latent and arrested cases than with improving and advancing cases.

DISCUSSION

Dr. H. C. Wood, Jr.: I am very much interested in Dr. Cohen's paper, but have nothing to say about it except a word of protest about his use of the word "autonomic." It is a term used, I think, with an unfortunate degree of ambiguity today. Apparently, Dr. Cohen used the term as synonymous with sympathetic; but, in this country at least, physiologists and pharmacologists divide the sympathetic system into two parts: the sympathetic proper, and the autonomic system. Most of the phenomena to which Dr. Cohen referred are vasomotor phenomena and the bloodvessels, with the exception of those of the face, are supplied from the sympathetic proper, not from the autonomic system. It would have been much more in accordance with common use of the word to have spoken of sympathetic phenomena rather than autonomic phenomena. How we can get migraine from either the sympathetic or autonomic system I do not know, because neither of the system of nerves has any connection with the nerves of sensation. The thyroid gland nerve supply is at present purely hypothesis.

APPENDIX

PROCEEDINGS

OF THE

SECTION ON OPHTHALMOLOGY

JANUARY TO DECEMBER, 1916

JANUARY

Previous to the stated business of the meeting there was placed on exhibition about two hundred water-color pictures of pathological lesions of the eye painted by Miss Margaretta Washington. In recognition of her excellent work the Section passed a vote of appreciation.

Pulsating Exophthalmos

Dr. William Zentmayer presented a patient with the following clinical history: D. F., aged twenty-one years, miner, was struck in the right occipital region by a mine car, on July 7, 1915. He was unconscious for five days. No operation was performed. On regaining consciousness the right eye was prominent and there was a buzzing noise in the right ear "which kept time with his heart." No diplopia, headache, or pain. The left side of his face was paralyzed. For the latter condition he has had vibratory massage and electric treatment, and to this he attributes the improvement that has taken place in this symptom. The left eye has been at no time involved.

R. E. proptosed 10 mm. The superior orbital sulcus is pushed forward by a soft mass in the orbit above the globe; ordinarily

this mass is more prominent to the temporal side, but when the lids are forcibly closed the swelling becomes more marked especially to the nasal side, and a thrill may then be detected over the mass. There is a swishing bruit synchronous with the heart systole. Pressure upon the carotid controls both the bruit and the proptosis. The external rectus muscle is paralyzed. The bulbar conjunctival vessels are greatly engorged. One large vessel extends from canthus to canthus. R. pupil, 3.5 mm. prompt to light; L. pupil, 2.5 mm. prompt to light. R. E., 20/20; L. E., 20/15. Ophthalmological examination showed in R. E. veins and arteries enlarged and somewhat tortuous, more particularly in the nasal branches. The venous blood current is very dark. The case is now in the hands of the surgeons, with a view of determining the proper surgical procedure.

Rupture of Eyeball by a Golf Ball, being a Demonstration of the Histology of the Enucleated Globe

Drs. G. E. de Schweinitz and H. M. How demonstrated the sections of an eye removed about three months after it had been ruptured by a hard-driven golf ball. At the time of examination the eyeball was moderately injected and the tension minus. Through the cornea were several small scattered infiltrates, and the iris was markedly atrophic and apparently pushed against the posterior surface of the cornea. At the upper corneoscleral junction there was a slight depression of the sclera and a bunch of prolapsed iris tissue protruding, behind and below which was a large yellowish-white exudation.

Microscopically it was found that both lips of the limbal wound presented advanced epithelial proliferation with an extension of the epithelial cells up and along either side of the protruding iris. The cataractous lens was adherent by its capsule to the posterior surface of the cornea, and on either side of the lens the greatly stretched and thinned iris was adherent. Although flattened the ciliary body preserved its structure in fair degree. The retina was greatly thickened, showing large spaces due to edema. Except

that the choroid was detached for a short distance posterior to the ciliary body its structure was fairly normal. The exudation in the anterior chamber was homogeneous in structure and contained cellular detritus.

Kerato-iritis in a Boy with Polyarthritis (Still's Disease), Being a Clinical and Pathological Report

Dr. G. E. de Schweinitz and Dr. H. W. How recorded the case history of a boy, aged five years, originally under the care of Dr. G. G. Davis in the orthopedic department of the University Hospital and later in the ophthalmic department of the same hospital, in whose family history tuberculosis on the father's side was the most important point. At the age of three years the child first developed arthritis of both knees. Two years later the left elbow, both wrists, and the joints of several fingers were involved, and slight ankylosis of the jaw was evident. Enlargement of the axillary, cervical, and inguinal glands was present. The condition appeared to correspond with the symptomatology of Still's disease, although subsequently the orthopedic department, in consultation with the children's department, expressed some doubt in regard to the exactness of this diagnosis, and was inclined to classify the condition as one of chronic polyarthritis. No good effect followed removal of the tonsils. Tests to determine the presence of tuberculosis were negative. The Wassermann test was thrice repeated with negative results and once with a delayed negative. About three years after the first arthritic process had begun, iritis of the right eye developed, which subsided under treatment, only a few posterior synechiæ remaining. Not so with the left eye, which, as the result of numerous relapses, passed into a condition of chronic iridocyclitis, or kerato-iridocyclitis, with infiltration of the cornea, especially in its lower part, associated with slight vascularization, filling of the anterior chamber with yellowish exudate, traversed with a band of hemorrhage. The constant exacerbations of cyclitis, uninfluenced by treatment, necessitated enucleation.

The microscopic examination revealed swollen and edematous corneal plates, with capillary vessels in the middle layers of the corneal stroma, an anterior chamber filled with an albuminous exudate, posterior to which, completely closing off the anterior chamber posteriorly there was a dense band of fibrous tissue, about two-thirds the thickness of the cornea, and somewhat resembling it in structure. This curtain-like band which extended from one angle of the anterior chamber almost to the other angle was somewhat adherent to the iris, although sharply demarcated from it in structure. The iris itself was densely infiltrated with small lymphoid cells, and throughout the stroma, but chiefly at its base, were numerous large plasma cells. The ciliary muscle was atrophic and contained large masses of round cells and lymphocytes. In the lens were masses of vesicular cells which appeared to correspond to the blasenzellen of Wedl. The band of fibrous tissue described crossing the anterior chamber corresponded in appearance almost exactly to the one illustrated in Fig. 191 in Parson's *Pathology of the Eye*, vol. i, Part I, p. 290.

Unilateral Pulsating Exophthalmos

Dr. G. Oram Ring exhibited a patient showing a very marked degree of unilateral pulsating exophthalmos.

The history of the case was one of unusual interest, inasmuch as the etiology was associated with a congenital growth situated at the upper and inner orbital margin, which was operated upon at the University Hospital by the late Professor William F. Norris thirty years ago, it being regarded as cyst of the orbit with supernumerary lid from the caruncle.

This was followed by a recurrence and second operation, which in turn resulted in a gradual orbital distention of vascular type, evidently emanating from the upper and inner orbital region and that of the caruncle, which in turn induced a pronounced and slowly progressive proptosis.

The exophthalmic eyeball remained during this prolonged

period, slightly functioning with the possibility of lid closure, until a recent influenzal attack caused a marked increase of symptoms with an exposure keratitis of the lower third of the cornea.

Miss H. H., aged thirty-two years, was admitted to the medical ward of the Episcopal Hospital, in the service of Dr. A. A. Stevens, last December, suffering from an acute influenzal infection which resulted in a pneumonia.

The personal history was unimportant, barring the existence at birth of a small growth at the upper and inner orbital margin, which showed a disposition to increase in size when she was two years of age, at which period it was removed as above recorded.

Mother living at seventy-two years of age, father died of thrombosis at sixty-eight, cardiovascular disease being present in each parent.

Upon transference to the eye ward the left eyeball was proptosed forward, downward, and outward, the pole of the cornea being 17 mm. farther forward than the normal or fellow-eye and 10 mm. below it.

The eyeball was greatly injected, its movements moderately well preserved, and the cornea at its lower third presented an ulcerated area of exposure keratitis, the upper border corresponding to the point of closure of the lid.

In the lens up and out was a triangular dense subcapsular opacity, from the bottom of which a spur passed into the lens substance. The edges of the opacity were eroded. The fundus, which was difficult to examine, showed areas of retinochoroiditis with extensive choroidal atrophy.

The forward swelling and distention of the lid area began 10 mm. above the brow and the measurement from this point to the edge of the lid was 40 mm. The veins of both lids were markedly dilated and the distance to the terminal of the swelling and edema of the lower lid from the point of beginning in the brow was 58 mm. Under the upper lid was an enormous blue-red swelling with a thickened fold of conjunctiva, growing less toward the outer canthus. A similar less marked condition of the conjunctiva and

vascular tissues existed beneath, with a symblepharon at about the inner third.

A serosanguineous fluid was more or less constantly oozing from the conjunctival vessels, and over the lid area a bruit could be distinctly heard.

In a good light fingers could be indistinctly outlined.

Since there was no history of traumatism other than that induced by the original operative intervention, the case was regarded as an aneurysmal dilatation of one of the branches of the internal carotid after it leaves the sphenoidal opening, probably secondary to the original operative procedure.

Dr. Ring said it was well known that pulsating exophthalmos may be an aneurysm of the ophthalmic artery either intra- or extra-orbital, an aneurysm of the internal carotid, or a so-called arteriovenous aneurysm involving both carotid and the cavernous sinus. Dilatation from the obstruction of the ophthalmic vein is also frequently in evidence. He thought it was not always possible to differentiate these conditions, and not absolutely necessary to do so from the viewpoint of the treatment if ligation of the carotid was decided upon.

Ligature of the common carotid was advised as a safer procedure than orbital intervention, an opinion concurred in by Dr. Thomas S. Neilson, senior surgeon to the hospital.

Ligation of Ophthalmic Vein for Pulsating Exophthalmos

Dr. F. Krauss described a method of ligating the ophthalmic vein in pulsating exophthalmos. The eyebrow having been shaved, an incision is made directly on the upper line of the eyebrow, extending from above the outer canthus to and along the side of the nose down to the periosteum. The periosteum is then gently separated until the whole contents of the orbit can be readily displaced intact. The pulsating vessel can now be located with the finger and its direction noted. An incision is made through the periosteum near the longitudinal axis of the vessel and a catgut suture introduced in a wide sweep by means of a pedicle needle.

Through-and-through sutures, including the periosteum, are used to close the wound. With a little care the entire mass of enlarged veins can be completely tied with very little or no danger of hemorrhage. Any growth on the inner half of the orbit can be readily reached in this way from behind the periosteum.

Traumatic Pulsating Exophthalmos; Ligation of Orbital Vein

Dr. de Schweinitz and Dr. Holloway exhibited a patient who had been admitted to Dr. Frazier's service at the University Hospital on November 26, 1914, with a fracture of the skull as the result of a motorcycle accident. On admission the right pupil was contracted and reacted sluggishly to light, the left pupil was dilated and did not react. As the patient's mental condition cleared he noted that the vision of the left eye was affected. On December 3, 1914, a left temporal decompression was done. Five days later examination of the eyes showed that the left eye was proptosed and displaced downward. There was some limitation in all rotations of the globe except downward. The temporal half of the disk was pale and the retinal veins were full-sized. The hand field was full. He could count fingers at one meter. The examination of the right eye was negative. About this time he developed a purulent middle-ear infection. He was discharged December 20, 1914.

On October 25, 1915, he came to the eye clinic stating that his eye condition had been gradually getting worse since his discharge. With Hertel's exophthalmometer, O. D. measured 11.5 and O. S. 20 mm. The distance from the midline of the nose to the centre of the right pupil measured 31 mm. and to the centre of the left pupil 33 mm. The left eye was 8 mm. below the right. Upon palpation pulsation could be felt which was increased by pressure upon the globe. A bruit could be heard over the supra-orbital region most marked internally. When lying on the left side subjective bruit was present. In the upper lid, beginning about the middle of the eyebrow and extending almost to the internal

canthus, there was a large venous ectasia over which a thrill and pulsation could be felt. A similar cherry-sized ectasia was present in the lower lid near the inner canthus. There was decided engorgement and tortuosity of the conjunctival and episcleral vessels. The disk showed some pallor, and plastered over the whole fundus were thin hemorrhages of varying sizes. The veins were full and tortuous, the arteries not reduced in caliber. The rotations of the globe appeared to be full aside from slight impairment of the outward rotation. The veins over the left frontal region were moderately engorged. The vision was 6/22. Right eye healthy.

On November 14, 1915, Dr. Frazier cut down on the upper venous ectasia and found that this was produced by a much engorged vein that was doubled on itself. This was traced far back into the orbit, after which it was excised between ligatures. Prior to the orbital procedure a ligature was thrown around the left common carotid artery but was not tied. The day following the operation the subjective bruit had disappeared. The course was uneventful and he was discharged from the hospital November 27, 1915. Microscopic examination of the excised vein showed that the walls contained a partially preserved lining. The remaining portion was extremely thin, and showed considerable blue-staining substance resembling mucoid material.

On January 14, 1916, he again returned to the eye clinic complaining of some aching pain above the globe, which was relieved by lying on the left side of the head. There was no subjective bruit. The site of the operative wound was clean and the ectasia below had disappeared. On deep pressure in the upper and inner portion of the orbit a faint pulsation could be felt. Displacement of the globe was the same as before the operation. While sitting no objective bruit could be heard, but this could be elicited when the patient stooped forward or was lying down, and when in the latter position a bruit de piaulement could be intermittently heard.

Dr. S. D. Risley urged the value of systematic compression of the carotids before ligation was undertaken as an aid to establishing

the collateral circulation. He related briefly the history of one case of aneurysm at the apex of the orbit where a cure was effected by compression alone. The exophthalmos, bruit, and pulsation were present. The bruit and pulsation ceased under pressure upon the carotid, but caused vertigo. This was repeated at short intervals many times during the first consultation. When seen a few days later the exophthalmos remained unchanged, but the pulsation and bruit were absent. Exploration showed an unyielding tumor at the apex of the orbit, approximately the size of a chestnut. The proptosis of the ball gradually but entirely disappeared. Dr. Risley thought that the stoppage of the blood stream through the aneurysmal dilatation had resulted in the formation of a clot, which later became complete.

Dr. J. B. Turner inquired of the members present what had been the after-results of tying the common carotid artery for pulsating exophthalmos. Recently he had a case in which the common carotid was clamped according to the method of Matas and the patient after four weeks was doing well. The case however was not one of exophthalmos, but of hemorrhage following a nasal operation. The advantage of clamping is that the clamp can be removed in forty-eight hours if necessary.

In conclusion, Dr. Holloway stated that while at times carotid ligation had its serious consequences, statistics showed that this operation was less apt to be followed by serious results in pulsating exophthalmos than when performed for other conditions.

He would like to suggest for Dr. Ring's consideration the advisability of gelatin injections, which he thought might be given a trial before resorting to more radical procedures.

Dr. Ring in closing the discussion referred to a case of exophthalmos treated about five months ago by ligation of the common carotid, with almost complete recession of the proptosed globe. In this case, however, the exophthalmos had existed for only about two months, and while very pronounced, was thought to have followed a traumatism through the os planum. Dr. Ring felt that the case of Dr. Turner was hardly germane to the discussion inasmuch as ligation was not for the condition of

exophthalmos but for excessive hemorrhages following excision of the middle turbinal. Dr. Ring expressed himself as being very willing to use the 2 per cent. solution of gelatin before adopting the more radical procedure.

FEBRUARY 18

Angioid Streaks in the Retina

Dr. Charles R. Heed presented a boy, aged eighteen years, showing angioid streaks. In each eye the media were clear, disk of a high color with margins well defined, and a choroidal ring all around. The retinal vessels were of good calibre. There were decided pigmentary changes of a granular form associated with a guttate choroiditis especially marked to the temporal side of the macula. Lying between the retinal and choroidal vessels were a number of streaks, of a dark brownish color, branching freely and apparently arising from an ill-defined pigmented ring surrounding the disk. They gave the impression of an anomalous vascular system. No hemorrhages were observed in either eye. Repeated Wassermann tests were negative and there was no tuberculosis in the immediate family.

Dr. Zentmayer, in discussing Dr. Heed's paper, said the point of greatest interest in such cases concerned the origin of the streaks. The one constantly associated condition was a choroiditis, usually located in the periphery and of a coarsely granular nature. Retinal hemorrhages have also been present in the majority of cases, but Dr. Zentmayer thought the view that they were in some way metamorphosed into these vessel-like bands was not in harmony with any other pathological change with which we were familiar. That a chronic inflammation of the choroid should lead to the development of new vessels was in keeping with the pathological findings in eyes removed for chronic inflammation of the uveal

tract, Lester having found such vessels anatomically in eyes removed for iridocyclitis. It is of interest that two observers arguing from the same premises should come to exactly opposite conclusions. In both Spicer's case and Dr. Zentmayer's there was an interruption in the course of one of the streaks by a small area of retinochoroiditis; an observation which Dr. Zentmayer believed argued for the vascular nature of the streaks and which Spicer believed indicated their pigment origin.

Mikulicz's Disease

Dr. Posey reported a case of symmetrical lymphomata of the lacrimal glands—Mikulicz's disease. The patient, a married colored woman, aged twenty-one years, had first noted the swellings over both eyes four weeks previous. There had been no pain or signs of inflammation. Wassermann + 4. Areas of dulness over the lungs. Positive von Pirquet. Hemoglobin, 77 per cent. Sublingual, thyroid, submaxillary, inguinal, and femoral glands enlarged. Five of six teeth decaying with gingivitis. Examination showed hypertrophy of both lacrimal glands with characteristic bloodhound appearance of eyes. No displacement of globes. Upward motions of globes somewhat restricted. No changes in eye-grounds. Patient receiving cod-liver oil, iron, bichloride of mercury, and general supportive treatment.

Dr. Posey referred briefly to another case seen by him years before, also occurring in a colored person, a girl, aged fifteen years. Both glands were affected. There was doubtless a tubercular element in this case also, as the left eye had been previously infected by phlyctenular ulcers. There was no apparent involvement of the glandular system elsewhere. When seen two years later the swelling in the glands had disappeared without treatment.

Dr. Posey said that this rare disease was first described by Mikulicz in 1888. In addition to the enlargement of the lacrimal glands a similar hypertrophy of the salivary glands is also usually present. The accessory lacrimal glands and the parotid and preauricular glands may also be similarly involved. Observers have

also noted enlargement of the sublingual glands and those of the palate. Nodule swellings have also been noted in the tip of the tongue. Ziegler noted enlarged tonsils in all three of his cases. The facial expression is characteristic, the drooping of the lids resembling those of a bloodhound. The lacrimal glands are tense and brawny and may be moved slightly under the skin. The disease may apparently occur at any age, the limits of recorded cases ranging from four to seventy years. Males and females are equally liable. The disease seems to have a marked predilection for the colored race. The course is essentially chronic, extending over months and years. The etiology is obscure. Although both of the cases observed by him were tubercular, Dr. Posey said that many cases observed by others have been free from this disease. To quote Ziegler's extensive monograph upon the subject, "The etiological factors which most writers have recorded in their cases are: (1) infection from buccal or conjunctival bacteria; (2) glandular irritation from some toxic agent in the blood or lymph stream causing lymphatic hyperplasia; (3) some idiopathic origin." Ziegler thinks the source of infection nasal and the means of transmission through the lymphatic capillaries.

Extirpation should only be performed when the integrity of the globe is threatened by pressure from the glands. Ziegler believes treatment should aim at improving lymphatic action and systemic oxidation. All obstruction in the upper respiratory passages should be removed by operation. Arsenic, the iodides, pilocarpin, thyroid extract, and *x*-rays have all proved of value in some cases.

Dr. Ziegler said that cases of Mikulicz's disease such as Dr. Posey had shown were rare, and that very few had been reported in this country, although he had been fortunate in treating and reporting three cases. Every case so far placed on record has been characterized mainly by negative findings. The most prominent feature has been the absence of any definite infection either in the glands or blood. The diagnosis of lymphosarcoma, syphilis, or tuberculous adenitis has been repeatedly proved wrong by the spontaneous involution of the affected glands. He had first called attention to the characteristic facial appearance of drooping

lids or "bloodhound face" in a paper before the American Ophthalmological Society in 1909. An interesting feature in connection with the disease is the retrogression or disappearance of all clinical signs in the presence of some acute intercurrent disease, with a return, however, of all symptoms of the primary trouble when the secondary lesion has subsided. Dr. Ziegler considered the disease to be a manifestation of a lymphoneurosis, causing glandular disturbance with perverted secretion of irritating chemicals that cause these glands to swell and form a true lymphoma. Whenever improvement had been observed it had followed increased oxidation, due to removal of the tonsils, the correction of intranasal obstruction and the administration of arsenic, the iodides, and thyroid extract.

Copper in the Vitreous

Dr. William M. Sweet showed a boy, aged five years, who came to the Wills Hospital three days after an injury of the left eye from the explosion of a dynamite cap, which was picked up in an abandoned mine. Several particles of copper penetrated the hand and leg, and were removed by the attending physician. At the time of examination there was a wound of the left lower eyelid and an apparent scleral wound of the eyeball slightly below the horizontal meridian on the nasal side, and about 4 mm. back of the limbus. The anterior segment of the eyeball was normal, the vitreous clear, and the fundus normal. Suspended in the vitreous a short distance back of the lens, slightly below the horizontal plane and about 1 mm. to the temporal side of the vertical plane, could be seen a thin, flat, silvery-like body, somewhat curved in its upper portion, which rotated on its vertical axis in movements of the eyeball. A number of sets of *x*-ray plates were made with the tube in various positions, but in no instance was there secured a shadow of a foreign body on any of the plates. During the four weeks that the case was under observation there was no congestion of the external tunics of the eye, the vitreous continued clear, and the body remained in its first position. Toward the end of this

period there was greater movement of the body in rotation of the eyeball, but it always returned to the former situation. Careful examination failed to show the shred or the fiber of the vitreous suspending the body. Although the foreign body did not show on the *x*-ray plates, the scleral wound and the appearance of the body seemed to point to it being of metallic nature, probably a thin fragment of copper. Dr. Sweet believed that by illumination of the body through the dilated pupil by a hand-mirror it would be possible to enter a pair of Mathieu forceps through a scleral opening and secure the metal, although in the event of failure it was recognized that removal of the eyeball would be necessary. The parents objected to an operation that might result in loss of the eye. Although there had been no reaction from the metal during the four weeks following the injury, Dr. Sweet believed that ultimately the vitreous would become clouded, so that attempts at extraction would then be futile and the eyeball would be lost.

Dr. Ziegler spoke in detail of a case somewhat similar to the one Dr. Sweet had shown which appeared at his clinic at the Wills Hospital several years ago. Both eyes were first operated on for traumatic cataract, after which a piece of copper could be seen in the vitreous of the right eye suspended by a slender thread. Such a thread is not visible in Dr. Sweet's case, but the movements of the foreign body denotes its probable presence. An incision was made in the sclera back of the equator, Liebreich forceps introduced and the piece of copper seized and removed. The operation was facilitated by the use of an electric headlight which enabled the operator to view the movements of the forceps through the dilated pupil. There was no reaction. Final corrected vision was $\frac{6}{9}$ in O. D. and $\frac{6}{6}$ in O. S., which is still maintained.

Dr. Hansell advised against any immediate operative interference, and gave as his reasons for postponing an attempt to extract the foreign substance:

1. The negative *x*-ray findings. He thought it possible that the object seen was not metal, for pieces of steel quite as minute have been diagnosed and located by radiographs.

2. The magnification of this body was very great, and that in reality it was extremely small.

3. Operation might be unsuccessful, attended with loss of vitreous and followed by retinal detachment.

4. The eye was absolutely free from irritation.

A Case of Jaw-winking

Dr. T. B. Holloway presented the case of a woman, aged forty-four years, who had recently come under his observation and who gave the following history:

The patient stated that at times there was distinct drooping of the right lid and that the lid retracted or, as she expressed it, winked when she ate. This feature was first noted by her mother when she was a nursing infant. While the palpebral fissures measured 9½ mm., she stated that at times the right upper lid drooped. The eyes fixed well in various positions except above the horizontal plane, when the right eye became divergent. There was a suggestion of lagging in the upward rotation of the right eye. Upon looking downward the right upper lid failed to follow the globe to the same extent as did the left upper lid. Upon attempts at chewing there was a retraction upward of the left upper lid which was accentuated upon lateral movements of the jaw. This retraction was excessively marked when the patient looked down, at which time there was an exposure of at least 4 mm. of sclera between the lid margin and the limbus of the cornea. The retraction was but slight when she looked upward. The diplopia fields indicated a paresis of the right superior rectus muscle. The pupils were equal and the eyes otherwise normal.

Dr. Holloway referred to the classification of Sinclair and other allied phenomena. The theories of Gunn, Bull, Harman, Wilson, Bruce, Pontico, and Lutz were referred to.

Dr. Posey referred briefly to a young Jewish girl shown at the Section some years before, in whom the associated movements in the right upper lid were provoked only by rotary movements of the jaw. The writer inclined, himself to the belief that these

anomalous movements were dependent upon some atavistic form of associated movements. In man, movements of the eyes and head were synchronous and associated. It is not unlikely that in some of the lower animals a corresponding association in movements between eye and mouth may be present.

MARCH 16

A Subretinal Mass

Dr. Charles R. Heed presented a boy, aged fifteen years, exhibiting a subretinal mass in the temporal field of the right eye. The ophthalmoscope disclosed an area of choroiditic atrophy encroaching upon the macula and in juxtaposition to the lower border a grayish circumscribed area protruding forward as evidenced by the coursing of the retinal vessels, the summit at least three diopters in front of the retinal plane. The clinical history, Wassermann and von Pirquet reactions gave no hint as to the etiological factor. The case was under personal observation for the last ten months, and during this period there had been no change in the scotoma outline in the field, no irritative symptoms, or evidence of anterior uveal involvement. A definite diagnosis had been deferred. An organized exudate resulting from the choroiditis had been considered possible.

Foreign Body in Sclera

Dr. S. Lewis Ziegler, in discussing the interesting case of copper in the eye presented by Dr. Sweet at the last meeting of the Section, referred to a patient with copper in the vitreous operated on by him about nine years ago. The young man applied for a re-test of his glasses during the past week and the ocular structures were found to be absolutely free from all signs of inflam-

matory disturbance or other sequela, and his vision was still maintained at a high standard, as a careful refraction gave him O. D. 20/40 for distance and J–1 for near, and O. S. 20/20 for distance and J–1 for near. The right eye was the one from which the piece of copper was extracted.

Dr. Ziegler presented another case of similar nature in which the foreign body appeared to be imbedded in the sclera of the right eye. The patient, R. M., was injured February 13, 1916, by a flying piece of metal as a result of the explosion of a cartridge in the coal which he was shoveling into a furnace.

O. D. examination showed moderate congestion of conjunctiva, slight ciliary injection more marked in lower half of globe, small elevation of sclera down and in about 6 mm. from the limbus. Skin of upper lid discolored. Small punctate subconjunctival hemorrhages at several points. Cornea clear, anterior chamber deep, pupil dilated under mydriatic. Color of iris normal. Floating opacities in vitreous due to probable hemorrhage. Fundus hazy, disk round, veins somewhat full. On the nasal side to the far anterior and a little below the horizontal meridian there was a large area of whitish exudate which seemed to be located in the structures of the ciliary body. The *x*-rays showed the foreign body 1 x ½ x ½ mm., 13 mm. back of the cornea, 4 mm. below the horizontal plane, and 12 mm. to the nasal side on the vertical plane, which would make it appear that the foreign body was just outside the sclera.

O. S. had small hordeolum in lower lid. Some conjunctival injection, but the structures of the globe were perfectly normal.

The vision of O. D. was 20/100 and of O. S. 20/100; tension normal. The patient was admitted to Wills Hospital on February 21, 1916, and an exploratory operation was performed two days later. The sclera and the tissues on the outside of the eye were examined with the greatest care, but there was not the slightest evidence of a foreign body protruding from the globe nor was there any metal located in the surrounding structures. The wound was closed and another *x*-ray photograph taken, which showed that the foreign body had not been disturbed, as the location was

exactly the same. It was decided, therefore, that as the foreign body was evidently located in the scleral tissue itself it should be allowed to remain and the patient kept under observation. The swollen area in the fundus over the site of the foreign body had greatly subsided and become somewhat atrophic. There had not at any time been an inflammatory disturbance in the eye and the corrected vision was as follows: O. D. S. − 1.25D. = C. − 1.50D. Ax. 60° = 20/50.

Membranous Occlusion of Pupil Following Secondary Infection. Treated with Phylacogen

Dr. S. Lewis Ziegler detailed the history of a case of infection following a cataract extraction in which there was a membranous occlusion of the pupil. In addition to local treatment nine injections of phylacogen, of 2 c.c. each, were given, which resulted in the complete absorption of the inflammatory exudate.

Tubercular Buphthalmos Treated with Phylacogen and Tuberculin

Dr. S. Lewis Ziegler presented a further report on a case of buphthalmos improved by injections of tuberculin and phylacogen which he had shown before the Section several months previously. The case had returned with a pronounced blepharitis marginalis and ulcerative keratitis, which had rapidly improved under tuberculin and phylacogen injections

Melanosarcoma of the Choroid of Unusual Density

Dr. G. E. de Schweinitz and Dr. How detailed the case history of a married woman, aged forty years, whose eye examination had revealed the presence of a chocolate-brown tumor, easily detected through the dilated pupil with oblique illumination. Her history contained no facts which seemed to have any bearing upon the presence of this growth, save only that she reported an

injury to this eye as having taken place some months prior to the discovery of the defective vision. After enucleation a dark brownish-black tumor, spheroidal in shape, which measured 10 mm. in diameter, was found to occupy approximately one-fourth of the vitreous chamber. It was in contact with the sclera externally and situated posteriorly to the ciliary body. The tumor was sharply circumscribed and apparently non-infiltrating and non-encapsulated. Owing to the extreme hardness of this tumor all sections were difficult, but its minute structure was studied from sections cut from a portion of the growth, and the tumor was found to be made up of small round cells of embryonal connective-tissue type supported by a loose stroma of young spindle cells. There were numerous good-sized bloodvessels, their walls being made up of tumor cells. The sclera was not invaded, and uniformly distributed through the growth were numerous dark brown finely granular pigment masses. The ciliary body was not involved and a study of a series of small sections demonstrated that the growth arose from the vascular layer of the choroid a short distance posterior to the base of the ciliary body.

Sarcoma of Iris

Dr. S. Lewis Ziegler presented a case of Sarcoma of the Iris. A. M., male, aged fifty-five years, applied at the Wills Hospital on February 23, 1916, complaining of poor vision in the left eye for the past six months and some redness which his family physician thought might be pink-eye, but had no pain. No limitation of ocular movements. The vision of O. D. was 20/30 and of O. S. 20/100. Family history good; no evidence of metastasis. There was no history of trauma. The right eye was normal.

O. S. showed congestion of the bulbar conjunctiva with some fulness of the anterior perforating vessels. No tenderness elicited on pressure. Cornea clear, pupil oval, axis 90°, 3 x 5 mm., reacted to light on the temporal side but not on the nasal. Anterior chamber shallow and entirely obliterated on the nasal side, due

to a tumor mass springing from the root of the iris and occupying an oval-shaped area 3 x 6 mm. The tumor was dark brown in color and sharply differentiated from the iris tissue, which was normal and bright blue. The tumor seemed wedge-shaped, the portion protruding from the posterior chamber into the pupillary area being thicker. The iris tissue near its attachment had undergone slight atrophy and was partly involved in the tumor itself.

O. D. cornea clear, pupil round, 2 mm., reacted to light and accommodation, iris blue. Ocular movements full.

The patient was admitted to the wards of the Wills Hospital and the left eye enucleated under ether. Horizontal section of the globe showed a large pigmented tumor located between the iris and lens on the nasal side and an equally large leukomatous mass extending subretinally from the ciliary region into the vitreous chamber. This was also surrounded by a deposit of pigment.

A Case of Extensive Blepharoplasty

Dr. William Campbell Posey presented the notes and photograph of a case of ectropion of the lower lid of the right eye following contraction from cicatrices occasioned by the removal of a neoplasm from the tissue near the alæ of the nose of the corresponding side some months previously. In consequence of the scarring, which was very extensive, the outer angle of the right side of the mouth was pulled markedly upward, giving rise to the condition designated as risus sardonicus. The removal of the neoplasm had at first been essayed by a charlatan, a cauterizing salve being employed for the purpose. This application, though destroying part of the periosteum covering the face of the superior maxillary, failed to eradicate the growth, requiring subsequent removal by dissection. A study of the case demonstrated the impossibility of correcting the deformity in the lid by grafts of any kind by reason of the thinness of the tissues under the lid and the roughened and irregular sublying bone. After freeing the tissues of the lid by an incision parallel to the lid and some

8 mm. below it, on a line with the superior part of the scar tissue, the gap formed by the dissection of the tissues in this position was filled in with a long pedicled flap taken from the skin of the forehead just above and parallel to the eyebrow. Layers of gauze smeared with White's bichloride salve were used as the dressing. Union was perfect, the stitches being removed in four days. As there was still some tension upon the lid, with the aid and counsel of Dr. Zentmayer and Dr. Moorhead, one of the surgeons of the Howard Hospital, where the operation was performed, the scar tissue was incised along its inferior border, in a line parallel with the mouth, and the upward drag upon the lip entirely relieved by a free dissection of the tissues. This gap was filled in by a second pedicled flap of considerable length taken from the neck and corresponding side of the cheek. The same dressing applied as in the first operation. Healing was prompt. Dr. Posey spoke of the value of a series of uninterrupted sutures to secure perfect coaptation of the flap to the neighboring tissues, after the flaps had been secured in their proper position by a few interrupted sutures. The photographs taken immediately after the operation delineate the size and position of the flaps. Dr. Posey was of the opinion that massage and time would do much to improve the present appearance of the patient.

A Case of Family Congenital Palsy of the External Rectus Muscle

Dr. William Zentmayer presented the history of a girl, aged five years, who was recently brought to the Wills Hospital with a complete paralysis of the left external rectus muscle. All other movements were full except the inward, which was excessive. There was slight narrowing of the palpebral fissure but no retraction of the globe in adduction. There were no fundus anomalies. H. = 5.D. Examination of the mother's eyes showed that she also had a complete paralysis of the left external rectus muscle. All other movements were full. There was no retraction in adduction. The fundus was normal. O.D., − 2.D.; O.S., − 4.D.

She stated that the squint in the child's eye was noted directly after birth, as had been the case also with herself. She further stated that her mother has had the same condition since birth. The mother, who was sixty-one years of age, was brought to the hospital and an examination showed exactly the same muscular condition as was present in daughter and granddaughter. In O. D., M. = 12.D.; O. S., M. = 3.D.

The grandmother was one of a family of seven children, six girls and one boy. She alone was affected. She is the mother of three children, two boys and one girl. The girl alone was affected. The patient is an only child.

Operative Results in Extensive Symblepharon

Dr. Frederick Krauss presented a case showing the operative results in extensive symblepharon, due to a burn. The cornea was covered in two-thirds of its extent by a thick fleshy mass continuous with the lower lid. The lower cul-de-sac was abolished except for a small area on either side. The mass was dissected from the cornea, a cul-de-sac made, flaps of conjunctiva were dissected from each side and sutured to cover the defect on the eyeball. The tarsal side was covered by a Wolff graft from behind the ear, and held in place by a broad superficial suture passed through the lower lid and tied over gauze on the forehead, making a deep sulcus in which the graft remained in position.

Nodular Iritis Accompanying a Polyarthritis of Undetermined Origin

Dr. H. Maxwell Langdon described the case of a boy, aged thirteen years, who was seized ten days after an illness, which was diagnosed as "pneumonia," with a severe pain in the left knee. Shortly afterward both hips were involved as well as the ankles and wrists. He was admitted to the Orthopædic Hospital, with great stiffness of the joints, tenderness, bed-sores on his back, and a temperature varying between 99° to 102° F. Three weeks

after admission he had a severe pain in O. D., with some ciliary injection, and there developed a small gray node at the upper inner pupillary margin, 1 mm. in diameter. In forty-eight hours it had tripled in size, and in another twenty-four had begun to send out streamers of soft gray exudate which in two days filled the entire upper, inner quadrant of the anterior chamber. The general diagnosis had been thought to be either an aberrant form of tubercular infection or a low-grade purulent organism. The serum from a tapped joint was clear and gave no culture growth. On the third day of the ocular condition, $\frac{1}{300}$ mgm. of T. R. was given with no effect save the temperature then remained between 99° and 100° F. In forty-eight hours an injection of $\frac{1}{300}$ mgm. was given, and again no change except the temperature stayed between 99° and 99.5° F. Another joint yielded a cloudy fluid with no growth, even on dextrose-ascites-agar anaërobic stabs. Two sets of diplococci were found in a smear.

A third T. R. ($\frac{1}{300}$ mgm.) was given, and after this there was noticeable absorption of the exudate, which in forty-eight hours had disappeared, leaving the node filling in the pupillary area. The iris did not dilate well under large doses of atropin, and the node had taken on a distinctly yellowish tinge, as though purulent, and light perception was lost. It seemed possible that the diplococci found were broken chains of streptococci.

APRIL 20

A Case of Microphthalmos with Orbital Cyst

Dr. William Zentmayer presented the case of E. R., aged seventeen years. He was born with the present condition of the left eye, except that the swelling about the eye has increased since the twelfth year. No other member of family was similarly affected.

There was a swelling of the lower lid, with ectropion. A tumor

mass could be felt through the lid about the size of a small walnut, and apparently globular. It seemed to be anchored toward the temporal side and was but slightly movable. There was ectropion of the lower lid of such a degree that the cul-de-sac was obliterated. The conjunctiva was thickened. In the upper inner portion of the orbit there was a small rudimentary globe. The cornea was about 4 mm. in diameter. The globe had limited movement in all directions but downward. *X*-rays did not show the cyst. In O. D. there was a partial coloboma of the optic nerve. The excavation involved all but a narrow rim of the papilla above, and at the lower margin the inferior artery and vein emerged through a narrow bridge of glial tissue. The vessels entered the disk at the upper inner third. The bottom of the excavation was −3D. Fundus level slightly H. Since the case was exhibited it has been operated upon and the diagnosis confirmed.

Neuroretinitis following Removal of Thyroid Gland

Dr. Frederick Krauss presented a case of bilateral neuroretinitis following the removal of a goitre from a twenty-four-year-old man. The patient claimed to have had perfect vision and good health previous to the operation. He had failure of vision and symptoms of tetany several days after the operation. It has been three months since the operation. There was marked choking of the disk, the swelling in the right eye being +4D., and in the left eye +5D. There were no hemorrhages. The vision was normal in the right eye and nearly so in the left. The field for white was heavily cut in both eyes, the color field only slightly cut. The patient was very nervous, irritable, and depressed on account of transient attacks of amblyopia. He improved considerably after the administration of thyroid extract, showing the toxic nature of the lesion.

Dr. de Schweinitz, discussing Dr. Krauss's case, inquired in regard to the character of the periods of temporary amblyopia, and referred to this important sign of increased intracranial tension

in brain tumor, due to the pressure of a distended ventricle upon the underlying chiasm.

Dr. Holloway stated that he felt sure Dr. Krauss must have considered the possibility of the operation interfering with the equilibrium of the internal secretions. He thought this etiological factor should be borne in mind, especially when we consider the intimate relation existing between the thyroid and the pituitary body, and the relation between the thyroid and the pancreas and adrenals. Still further, a functional relation existing between the parathyroids and thyroids and the kidneys has been shown experimentally.

Ligation of the Ophthalmic Vein for Exophthalmos

Dr. Frederick Krauss exhibited a patient who had been operated successfully for exophthalmos due to orbital aneurysm. The primary operation had been ligation of the ophthalmic vein by the supra-orbital route. The pulsation and bruit then disappeared for two weeks, following which there was recurrence of the pulsations up and in from its former location. The exophthalmos was diminished markedly.

There were frequent attacks of bleeding from a small sinus in the upper inner angle of the orbit, apparently occurring and stopping without cause, for which the common carotid was tied.

The blood-pressure was reduced from 215 mm. to 150 mm., probably due to the bleeding. There were no complications. The exophthalmos had disappeared, and there was no pulsation or bruit. The ocular movements were good.

Disseminated Choroiditis

Dr. T. B. Holloway exhibited a boy, aged thirteen years, who was first seen in Dr. de Schweinitz's service at the University Hospital on March 13, 1916, at which time his eyes showed the following conditions:

The vision of the right eye was 6/12 and of the left 6/15. In

the right eye there were a few vitreous opacities, the disk, of rather mellow appearance, was pinkish yellow in tint. Studding the choroid were innumerable lesions, varying in size from a split pea to half the disk diameter. Many of these presented a grayish-white, cotton-like appearance, and were surmounted centrally or eccentrically by a ring of pigment having an apparent diameter of about 4 mm., the circumference of the lesion not being pigmented. In a few instances the pigmentation consisted of a solid clump about the same size as the rings. Some of the lesions were of a yellowish tint, and one near the macula was without pigmentation, whitish in color, and surrounded by a narrow yellowish areola. In a few instances the retinal vessels were partially covered by pigment. In the left eye there were a few vitreous opacities, with a low-grade neuritis, a diffuse superficial retinochoroiditis, most preceptible in the macular region, and in the periphery there were a few vague choroidal foci without pigmentation. General examination revealed slight enlargement of the cervical lymph glands, some enlargement of the tonsils, and a suggestion of a rachitic rosary. The Wassermann test was positive.

Dr. Holloway stated that he had never seen a case before quite as striking as the one under discussion. He believed the condition presented various stages of an active process depending upon hereditary lues.

Pulsation of the Retinal Arteries

Dr. T. B. Holloway stated that the two patients showing pulsation of the retinal arteries from cardiac and vascular disease were exhibited because he thought that ophthalmologists saw fewer instances of this phenomenon than was commonly believed. He stated this condition could be beautifully studied by means of the Gullstrand ophthalmoscope, as was done in one of these patients. He referred to the various conditions under which pulsation of the retinal arteries could be observed, as described by Parsons and Gowers.

The first case had been referred to Dr. Sinkler's clinic at the Orthopædic Hospital and Infirmary for Nervous Diseases by Dr. Hooker. There existed an aneurysm and a double aortic murmur as well as a left-sided hemoplegia. The Wassermann test was positive. The second patient was a young girl who came under observation in Dr. de Schweinitz's service at the University Hospital, and had mitral and aortic disease dependent upon rheumatism.

Dr. Zentmayer said that sometimes it was very difficult to be sure of the presence of an arterial pulse. Haab points out that it is important that the patient, the ophthalmoscope, and the observer's eye be absolutely steady, as the gentle pulsatory movement of the patient's body or in the arm of the observer may imitate a slight pulsation. He advises that the patient be seated with the arms upon a table and that the surgeon also have a rest for his arm. He thinks that through the medium of "autosuggestion" there is also danger of seeing pulsation when it is not present. Dr. Zentmayer recalled a case seen in consultation in which a pinpoint corneal opacity gave rise to the impression of an arterial pulse. The slightest movement of the mirror brought the artery alternately into view or obscured it, and it was only after careful study that the explanation of the phenomenon was reached.

In closing, Dr. Holloway stated that the phenomenon that Dr. Zentmayer referred to was observed in the first patient, and when examined it had been necessary for him to support the arm, as for some reason there had been a distinct diminution in the amount of pulsation. He regretted to say that he had not examined the cornea with a loupe for a possible faint nebula.

Extensive Epithelioma of the Lids Treated by Curettement and Radium

Dr. T. B. Holloway exhibited an Italian, aged about fifty years, who was first seen at the Polyclinic Hospital on March 25, 1915. The patient had a large epithelioma about the internal canthus,

involving the inner portion of both lids. It began as a small papule on the lower lid four years before, and had not received treatment for three years. Beginning at the middle of the lower lid there was an ulcerating area, the size of a five-cent piece, that extended inward to the inner canthus. A somewhat similar but smaller area involved the inner portion of the upper lid, the two being connected by a narrower tract extending deeply at the internal canthus, involving the lacrimal sac. From the inner margin of the lower lid a flap-like mass of epitheliomatous tissue extended upward, partially covering the inner portion of the globe. The patient was not seen again until four weeks later, when the lesion had much enlarged, and at which time he consented to operation. An extensive curettement of the whole epitheliomatous area was done, necessitating deep excavation into the orbit. After the application of pure carbolic acid a moist dressing was applied. The result of this was most satisfactory, the upper area filling in with a barely perceptible scar. Three months later a recurrence at the lower orbital margin necessitated another curettement, and owing to a recurrence at the outer portion of the lower lid a third curettement was done on February 23. Since the second operation three applications of radium have been made by Dr. Pancoast, with little or no reaction and very satisfactory results. At the present time the eye is quiet, and only about 5 mm. of the outer extremity of the lower lid remains.

Dr. Zentmayer said that in his experience the results obtained from the "desiccation treatment," as done by Dr. W. L. Clark, had been so satisfactory from every point of view that other methods of treatment, including plastic operation, had been abandoned except when the destruction of tissue was extensive at the time the case was first seen. He believed his experience was in agreement with that of his colleagues at Wills Hospital.

Dr. de Schweinitz, discussing Dr. Holloway's paper, described a patient with epithelioma that had destroyed partly the outer commissure, the lower lid, and had extended inward at the inner angle of the orbit. Pain was severe. The eyeball, which was beginning in its lower area to become epitheliomatous, was enu-

cleated, but complete evisceration was not performed; only the evident epitheliomatous tissues were excised. One treatment of radium (50 mg.) for four hours was followed by very rapid cicatrization and complete relief of pain. A few weeks later a small area of breaking down tissue at the inner and upper angle of the orbit occurred, quickly relieved by a second application of radium. Some months have now elapsed and there has been no recurrence.

Case of Round-celled Sarcoma of the Choroid

Dr. Edward A. Shumway reported the case of a man, aged fifty-two years, in whom the first microscopic sections showed an isolated round nodule, lying between the thickened, detached retina and the lens, composed of non-pigmented round cells, of the same size as the nuclear cells of the retina. There was necrosis of a considerable part of the tumor, leaving well-staining mantles of cells surrounding the bloodvessels as in glioma, and the growth had the appearance of having sprung from the retina. Further sections of the other half of the eyeball, however, showed the tumor to have sprung from the choroid, and to be in reality a round-celled leukosarcoma of this tunic.

A Case of Pituitary Body Struma, with Special Reference to the Visual Field Phenomena and the Effects of Organotherapy

Dr. G. E. de Schweinitz and Dr. H. W. How detailed the clinical history of an unmarried woman, aged fifty-one years, whose occupation was that of a tuberculosis dispensary nurse. There is nothing of importance in her family or previous medical history, except that after a prolonged siege of nursing she broke down nervously and lost flesh rapidly, as much as thirty pounds. Later, after retiring from this work, the lost weight was regained. Menstruation was established at fourteen years, and always occurred in association with intense pain. Since her forty-ninth year there has been no regular period, and none during the last year. Always,

even from early life, she had intense headaches, sometimes neuralgic in type, unaffected by any ordinary treatment. She has had much rheumatism. Her mother was a victim of rheumatoid arthritis. In childhood, convulsions occasionally occurred, and in recent times much numbness in the hands, which was present in all of the fingers and in the thumb of the left hand.

Her eyes had been under observation for fifteen years, and during that period of time had never shown any pathological conditions until early in June, 1914, headaches at that time being very severe, when she complained of blurred vision of the right eye, which previously had always been normal. Examination showed O. D., 6/60; O. S., 6/9; marked pallor of the optic disks. Visual field examination demonstrated bitemporal hemianopic scotomas, and *x*-ray examination, the typical picture of enlarged sella, suggesting the presence of pituitary body neoplasm or struma. Gradually the vision of the right eye began to improve, but the vision of the left eye deteriorated until nine months after she first came under observation the visual power had dropped to 4/150. The visual power of the right eye never dropped below 6/60. The visual field defect of each eye continued to increase until there was well-marked bitemporal hemianopsia.

The patient declined operative interference, and was placed upon tablets of thyroid extract and pituitary body extract, $2\frac{1}{2}$ grains each, and had taken at the time of the report about 1400 of these tablets, altogether, therefore, about 7000 grains of the medicine from about July 1, 1914, until February 24, 1916. A series of visual charts covering this period of time were exhibited, showing the gradual development of bitemporal hemianopsia from paracentral scotomas, and the gradual return of the visual field, chiefly from below upward, until the visual fields were restored practically to their normal extent, and the vision of the right eye was 6/5 and that of the left eye 6/12. Headaches and other symptoms had practically entirely disappeared.

The authors referred to another case, which one of them (Dr. de Schweinitz) had already placed upon record, namely, that of a patient, aged thirty-nine years, with tumor of the pituitary

body, as demonstrated by the *x*-rays, with complete blindness lasting twelve days in the right and six weeks in the left eye, with complete restoration of the vision of the right eye and nearly complete restoration of the vision of the left eye, under the influence of large doses of thyroid extract associated with inunctions of mercury. Whether in the last-named case the mercury had any influence or not was uncertain. Syphilis was not present; at least the tests failed to show its existence. In the first case, however, the good result followed the use of thyroid and pituitary body extract without the addition of the mercurial treatment.

Dr. Holloway stated that he recalled very well the two most interesting cases that Dr. de Schweinitz had referred to, and was quite in accord with him in reference to the importance of this early obscured or hazy vision. He then referred to a patient in his own practice in which this was the only symptom complained of, and the fields showed characteristic changes for colors in the upper and outer quadrant, which was more of a hemianopic than quadrant defect, with a retained island for blue in the left field, which subsequently disappeared. Later an island for blue developed in the right field; whether this was an indication of a partial return of the field it was too early to say. He thought there were now enough cases on record to justify the routine use of organotherapy in selected cases. He then referred to persistent photophobia in a case of pituitary body disease which had remained constant even after several operative procedures. It was interesting to note that in this case there never had been any intraocular changes aside from hyperemic disks and full retinal veins. There were no field changes aside from slight contraction of the form and color fields. Dr. Landon, who performed the autopsy on this patient, found that the chiasm was lying much farther back than was normal, and that the optic nerves were not apparently attenuated. This case belonged to those few cases on record in which the growth was anterior to the chiasm.

Orbital Cellulitis with Optic Neuritis

Dr. Ernest B. Mongel (by invitation) reported the case of a young man, aged twenty-three years, who had an attack of influenza February 5, 1916. Four days later his right eye began to trouble him, which, according to the patient's description, was a catarrhal conjunctivitis. The condition became progressively worse and he was sent to Philadelphia and was admitted in Dr. S. D. Risley's service at the Wills Hospital on February 21, 1916. Vision on admission to hospital, R = 20/200, L = 20/20. In the right eye there was intense chemosis and edema of the conjunctiva of both eyelids, and thick folds of swollen and edematous conjunctiva of the lower half of the globe filled in and protruded from the interpalpebral fissure. All the tissues surrounding the eye were hard. There was a proptosis down and out of the right eyeball about 10 mm. The lids could not be everted. There was an absolute suspension of all the ocular movements. The cornea was clear and the anterior chamber of normal depth. Iris was negative. Pupils equal round 4 mm. respond promptly to light and accommodation. T = normal.

Media clear. Fundus: vessels full. Veins dilated and dark, and somewhat tortuous. Nerve head hyperemic, edges very blurred, margins swollen about 6D.; typical picture of optic neuritis.

Left Eye. Anterior segment normal. Media clear. Fundus negative. The patient was put at absolute rest in bed and given free elimination and tonics. Constant cold compresses to eye, and atropine locally to eye. After the course of a few days, during which time there was very little pain and tenderness, and less than 1° rise of temperature, the swelling of the tissues gradually lessened, as did the swelling of the nerve. This went on for ten days, when it became evident that the tissues were breaking down. Pus formation set in and signs of pointing showed in the lower lid near inner canthus. In this location a long slender knife was thrust in along the floor of the orbit and directed along toward nasal side, and the profuse quantity of pus evacuated. After this

the cavity was thoroughly cleansed and drainage tube inserted and hot alkaline stupes applied. After a few days the swelling of tissues was almost gone. Ocular muscles regained power, and all ocular movements became normal. Nerve head and general fundus also gradually recovered normal condition, and patient left hospital with a correcting glass, which gave him 20/20 vision in each eye.

The most plausible explanation in this case is that the infection travelled from the ethmoid cells through the nasal fossæ, but since the *x*-ray of the sinuses was negative the only explanation could be metastasis or endogenous infection from grippe.

Optic Neuritis following Influenza

Man, aged sixty-seven years. Had an attack of grippe. After about one week he noticed that his sight was becoming very dim. After about two weeks of the beginning of illness he was admitted to the Wills Hospital in a very debilitated and emaciated condition; so weak that he could hardly stand on his feet. He looked twenty years older than his age stated.

Vision on admission: O.D. = blind; O.S. = perception of light. Anterior ocular segments normal. Media O_2 = slight haziness of both lenses. Fundi showed optic nerve heads entirely obliterated, only by the convergence of the vessels could they be located. Swelling about 5 to 6D. Very hyperemic. Veins full, dark, and slightly tortuous, surrounding retina somewhat clouded and infiltrated, but no hemorrhages. Urine negative. Blood-pressure: S. = 110; D. = 60. Subnormal temperature. *X*-ray examination negative.

Patient was ordered absolute rest in bed. Eliminative and tonic treatment was ordered with nutritious diet. This was continued for about three weeks when the patient became very homesick and at his own request left the hospital with his physical condition much improved and the vision in O.D., light perception? O.S., counted fingers at 4 feet. During this period frequent examinations of fundus showed decrease in swelling of nerve heads and retinal edema.

OCTOBER 19

Orbital Tumor

Dr. Wm. Campbell Posey exhibited a case of orbital tumor, possibly an osteoma, growing from the roof of the right orbit. The patient, a male, age nineteen years, denied traumatism or other probable cause to account for the origin of the neoplasm. The growth first manifested itself in a drooping of the lid about eight or nine years previously, the swelling in the orbit coming on several years later. At first double vision was present, but after several months this troublesome symptom disappeared. The patient had always enjoyed excellent health and his family antecedents were good. Examination revealed a marked disfigurement of the face. The forehead is very prominent, this being especially marked on the right side. The right eye was much lower than its fellow and somewhat proptosed. Palpation revealed a hard mass just below and confluent with the supra-orbital rim of the right orbit. The mass was slightly bosselated and did not pit on the firmest pressure. It merged with the inner wall of the orbit, and its external border, which presented the same characteristics as the body of the mass, terminated just within the outer margin of the brow. The upper lid partially covered the right eye and could not be raised voluntarily. Upward motion of the globe was also lost, though the other movements were unimpeded. The fundus was healthy, the optic nerve showing no alterations. Uncorrected vision equaled $\frac{5}{10}$ in the right eye and $\frac{5}{5}$ in the left. There were no signs of inflammation about the eye or orbit, and the patient asserted that this had been the case since the growth was first noticed. At no time had he suffered any pain. As the patient had only presented himself on the afternoon of his exhibition before the Section, there had been no opportunity for *x*-ray or other studies. Dr. Posey thought the growth, in all probability, was an osteoma, and said that he proposed to extirpate it through a large incision under the brow.

Unusual Form of Retinal Degeneration

Dr. William Zentmayer presented the case of A. B., a girl, aged nine years, who was sent to Wills Hospital because of poor vision which could not be improved by glasses. V. each eye = $\frac{6}{60}$.

The ophthalmoscope showed in each eye, throughout the retina but much more marked along the course of the temporal vessels and in the macula region, disciform yellowish-white lesions averaging in size about $\frac{1}{3}$ d.d. Along the vessels they were discrete, but in the macular region they were confluent. Both nerve heads showed a low-grade optic atrophy. The child was well nourished, of good mentality. There was neither consanguinity nor heredity. v. Pirquet test was negative. Wassermann positive. The visual fields showed moderate concentric contraction.

Three Cases of Ocular Injury

Dr. Howard F. Hansell reported briefly the history of three cases of traumatism that presented unusual features:

Case I.—This was the case of a young man who was employed in the powder works at Hopewell, Va. He, in company with an older man, was approaching a large cylinder in the second-story room of one of the factories. As they came within a few feet of it there was a violent explosion, and together they were blown out through a door and over a railing and landed on the ground. The older man died within an hour. Of seven men who had been working in the room three were killed. The wall of the building was partly wrecked by the pieces of the exploded cylinder.

My patient believed his eyes were blown out. He was treated for two days in the hospital at Hopewell and then brought to Philadelphia and admitted to the Jefferson Hospital. He had intense photophobia; he was nervous and apprehensive, and feared permanent blindness. To my astonishment I found his eyes to be absolutely uninjured and not a bone in his body broken. He left the hospital and resumed his work the next day.

Case II.—R. was struck in the left eye with a wad of paper thrown with force by a fellow workman. He noticed a few days later declining vision without pain or signs of inflammation. The ophthalmoscope showed a patch of retinochoroiditis twice the size of the disk and fine dense vitreous opacities. In his case neither of the usual traumatic lesions—namely, dislocation of the lens or rupture of the choroid—were present. Under active diaphoresis vision improved to $\frac{20}{30}$, the chorioretinal patch diminished to one-half its previous size, and the opacities in the vitreous were partly absorbed.

Case III.—A physician about to operate received a splash of chloroform in each eye. For two days he was able to continue his professional work but at the end of that time intense pain commenced and continued without interruption for thirty-six hours. Sleep was possible only after the administration of large doses of morphia. Three days later he was admitted to the Jefferson Hospital after a painful journey of twenty-four hours. Both cornea were superficially infiltrated and a part of the epithelium destroyed. The conjunctivæ were intensely inflamed. The palpebral surface of each upper lid presented the appearance of subacute trachoma. The subconjunctival tissue was irregularly and unequally infiltrated with fluid and solid exudation as though the conjunctiva was granular. Bacteriological examination was negative. Under soothing local and general treatment he recovered perfectly in a few days.

Eskimo Snow-blindness and Goggles

Dr. Judson Daland (by invitation) stated that among approximately 3000 Eskimos living on the coast of Alaska, Siberia and the Islands of the Bering Sea and Arctic Ocean, the common ocular diseases were conjunctivitis, cataract, trachoma and snow-blindness. Notwithstanding the belief of certain authors that the Eskimo possesses relative immunity from snow-blindness, observations show that they are as susceptible as the Caucasian, and that one attack predisposes to another. Experience has taught

them the necessity of protection from snow-blindness; and necessity caused the invention of snow goggles made of wood. The Eskimo recognizes that snow-blindness occurs, not only on sunny but also on cloudy days, and he therefore insists upon the importance of wearing goggles on cloudy days.

Conjunctivitis and snow-blindness are usually associated with corneal erosions, choroiditis and retinitis.

The ultraviolet rays cause conjunctivitis, corneal erosions and possibly chorioretinitis.

Snow-blindness occurs in animals.

Dr. Posey said that he was under the impression that Peary had worn smoked glasses on his trip to the Pole, and that Shackelton had employed Euphos lenses. He had never seen a case of snow-blindness himself, but related an experience of his own while mountaineering at a height of over 10,000 feet in a thick fog, when both he and two comrades were startled by perceiving numerous white and colored spots dancing before their eyes and projected upon the clouds which surrounded them. He had thought at first that the appearance might have been due, at least in one of his companions who had a rather weak heart, to circulatory disturbances, from the altitude and strain of climbing, but as he himself and his other friend had never suffered from any such complications, he became convinced that the visual phenomena must have been attributable to causes which he could not explain.

Mikulicz's Disease

Dr. Posey also reported and showed a photograph of a case of Mikulicz's disease. The patient, a married colored woman, aged twenty-one years, stated that she had first noticed a swelling of the right half of the face a year previously, the swelling in the lids appearing three months subsequently. No other morbid phenomena presented themselves, though the swelling in the lids had slowly increased until the present time. She averred that with the exception of an attack of rheumatism in the left elbow five years previously, she had always had good health. Although

venereal taint was denied, Wassermann equalled +3. Examination showed characteristic swelling of lids, occasioned by the much enlarged lacrimal glands. Both parotid glands also enlarged, more so on the right side. Right pre-auricular gland palpable. Both submaxillary glands slightly enlarged. Epitrochlear and inguinal glands of right side palpable. Physical examination otherwise negative, with the exception of decay of three molar teeth. With the exception of some restriction in motion upward in each eye, the eyeballs themselves were unaffected. Dr. Posey stated that the strongly positive Wassermann suggested syphilis as the causal element, and it was his intention to begin a course of mercury at once.

NOVEMBER 16

A Case of Increasing Refraction following Increased Tension of the Globe

Dr. S. D. Risley presented a somewhat detailed history of a patient, now aged seventy-one years, who had for many years worn glasses correcting hypermetropic astigmatism. Later she became the victim of a mild form of arthritis deformans, with frequently repeated acute exacerbations in one or more joints. These attacks were often associated with painful eyes and with transient impairment of vision. The convex glasses were then rejected, and when first seen by Dr. Risley she had marked increase of tension of the right eye, with field contracted to near the fixation-point, a shallow anterior chamber, and vision greatly reduced. Even with the glass which she was then wearing, −2.00, vision was 1/4+. After a year and a half, characterized by numerous exacerbations of injection and increased tension, the hardness of both eyes almost suddenly subsided, the pain disappeared, anterior chambers more nearly normal, but myopia had increased to −4.D. in the right eye and −2.25 in the left, with vision 6/xv in the right

and 6/IX in the left. This change was accompanied by an obvious thinning in the anterior ciliary region and a groove-like formation surrounding the corneæ just back of the limbus, with tension normal in both eyes. There were no demonstrable changes in the crystalline lens to account for the increased refraction.

A Case of Recurring Neuro-epithelioma

Dr. G. Oram Ring exhibited a case of extensive recurrent neuro-epithelioma with the following history:

Esther K., a girl aged eight, was seen at the Protestant Episcopal Hospital January 7, 1916. The child was struck in the left eye three weeks before visiting the clinic: vision was reduced to 12/200; eyeball white; iris responsive but tremulous. The lens, which was completely dislocated to the nasal side just back of the ciliary body, was partly covered with exudation and two small hemorrhages. The lower half of the retina was detached; the irregularity of outlines suggesting the presence of a solid growth. There were likewise two irregular areas of glistening white degeneration up and out. Tension normal. No enlarged capillary vessels. Advice given; immediate admission to the hospital for further study and operation. The diagnosis: Lenticular dislocation (traumatic) with retinal glioma.

The child disappeared from observation at once and was not seen for nine months, during which period another dispensary service had been visited where the growth was regarded at first as a pseudoglioma. Enucleation was said to have been advised within two months from the date of the first visit.

On August 8 the child was again struck while at play by a little companion which resulted in a rupture of the sclera.

Enucleation was then performed at the institution above referred to but the enucleated eye unfortunately was not examined.

The child was again seen at the Protestant Episcopal Hospital about the middle of October, with an extensive recurrent growth quite two-thirds the size of a base-ball, with infiltration of the lids and temple.

Examination of the sinuses was thought to be negative but the lymphatic glands of the neck were somewhat enlarged. General examination otherwise negative. Transillumination at original visit to the dispensary was not satisfactory because of irritability of the child.

At the time of exhibition there projected from the orbit for a distance of 10 cm. a very extensive fungoid mass 22 cm. in circumference which was regarded as definitely neuro-epithelioma-tous in character and inoperable.

Three days before exhibition the enucleated eye was secured and an immediate examination by Dr. C. Y. White, director of the Episcopal Hospital Laboratory, confirmed the diagnosis.

Emphasis was placed upon the trauma and upon the added malignant disease as nullifying the usual diagnostic value of a shallow anterior chamber in malignant disease and a deepened one in vitreous exudation; the case showing a normal chamber.

Reference was made to the exhaustive studies of Wintersteiner and Flexner, as pointing to the origin of such growths in the neuro-epithelium of the retina.

The three varieties of neuro-epithelioma, exophytum, diffusum and endophytum were described and the dictum of immediate excision with prompt microscopic study to be followed by immediate orbital exenteration if the infiltration posterior to the nerve indicated the further presence of malignant disease was emphasized. It was pointed out that if early diagnosis is made and radical operation done, about 16 per cent. of such cases can be saved. Necessity for differential diagnosis in ten different ocular conditions was dwelt upon.

Cranial Deformity with Optic Atrophy; Opticociliary Vein in the Left Eye

Dr. T. B. Holloway exhibited a young man with the following history:

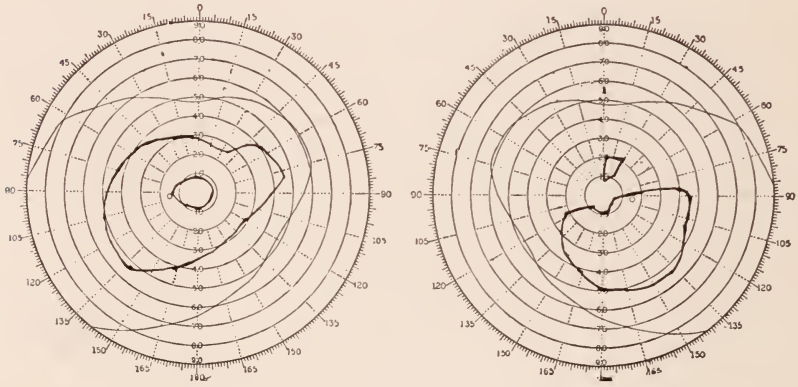
J. L., aged nineteen years, Hebrew. First seen in September, 1916, when he came to the Polyclinic Hospital desiring an opera-

tion for a divergent squint. He had poor vision as long as he can remember. He had been subject to convulsions since one year old, but during the past year the attacks had been less severe and he did not fall but became "dizzy" for about three minutes. His mother was dead, the result of pneumonia complicating labor. The father, three brothers, and three sisters were living and well. No brothers or sisters dead. He denied venereal disease, and

aside from the convulsions had never been ill. He was 5 feet 1 inch in height, and weighed 125 pounds. Neurological and medical examination failed to reveal any organic trouble. Nose and throat examination revealed a deviation of the nasal septum to the left side. No abnormalities aside from extra canine teeth. The head was not of true tower skull type, the deformity not towering anteriorly. The Wassermann test was weakly positive.

Skull measurements: Bitemporal, 117 mm.; stephanion diameter, 130 mm.; maximum transverse, 137 mm.; anteroposterior, 186 mm.; circumference, 524 mm.; biauricular curvature, 332 mm.; cephalic index, 73.6.

Both eyes were prominent: the right eye measured 22.5 the left 22 mm. with Hertel's exophthalmometer. There was a divergent squint of the right eye measuring 40 degrees on the perimeter. There were no extra-ocular palsies. Upon attempts to fix a near object the right eye remained divergent, but both pupils reacted with a well-marked contraction; both reacted well to direct and indirect light. Each disk showed a well-advanced atrophy with



but slight reduction in the caliber of the retinal vessels. On the left disk a small vessel entered the inferior temporal vein between its exit from the papilla and the disk margin. It described an S-shaped course and disappeared at the nasal margin of the disk into the choroid, where it could be followed for a millimeter or two. In color it corresponded to an artery. In appearance it corresponded to the description given by Shoemaker, *Annals of Oph.*, 1909, xviii, p. 241. Each eye was of the compound myopic astigmatic type. V. O. D. fingers 15 inches; V. O. S. 6/15.

Dr. Holloway referred to the various theories concerning the etiology of the cranial deformity as well as the optic atrophy. In exhibiting the stereoscopic *x*-ray plates made by Dr. H. K.

Pancoast he commented upon the enlargement and displacement of the sella turcica and the massive bony development of the face and thought it possible that disturbance in the function of the pituitary body might be responsible for the latter. With the right eye there was loss of the macular field and of the upper half of the field except a small isolated patch. He thought this might be due to some constriction or pressure about the optic foramen such as might occur in an exaggeration of the condition found and described by Behr.

Dr. Zentmayer asked whether Dr. Holloway had found in literature any reference to glandular feeding in these cases. The results secured from such therapy might have some bearing on the relation of pituitary disease to this condition.

Hyaline Bodies on Optic Nerve Head

Dr. Edward A. Shumway reported a case showing unusual development of hyaline bodies on the optic nerve head. The patient was a young man, aged twenty-one years, whose vision had been very poor since early childhood. The margins of each optic nerve were covered by extensive clusters of the hyaline bodies, which were peculiarly wax-like and glistening in appearance. The macular region in each eye was the seat of wide-spread superficial disturbance of the choroid and retina, so that vision was reduced to 6/30. The retinal vessels were reduced in caliber and the visual fields were contracted, especially for color. There was achromatopsia for green. Dr. Shumway thought the macular lesion might be considered a pigmentary degeneration of the retina without pigmentation. The two conditions had been reported together in a number of instances. He referred to the blurring of the nerve heads, usually present, and said he believed all cases of "pseudoneuritis" should be watched from time to time, as he had known of two cases, which for a long time were considered "pseudoneuritis," in which the condition had developed into choked disk, and upon operation brain tumor had been found.

Dr. Holloway stated he had seen but one other case in which the lesions were as marked as in the patient described by Dr. Shumway. He had observed the presence of hyaline bodies in cases of retinitis pigmentosa and referred to one patient who had been under observation at least six years, during which time the lesions had increased in numbers and were now quite extensive, but not as marked as in Dr. Shumway's patient.

Dr. Zentmayer said the presence of hyaline bodies on the papilla in pigmentary degeneration of the retina cannot be of so rare occurrence. He had seen 3 such instances in his own limited experience with this affection. Some years ago he presented at the section a patient with marked hyaline depositions on the papilla associated with hyaline changes in the retina and said that the case suggested that in some cases at least there might be a relation between the two conditions.

The Ophthalmologic Aspects of the European War as Seen in the American Ambulance of Paris

Dr. Hunter W. Scarlett stated that the peculiar nature of trench fighting produced so many head and face wounds that the number of eye cases was necessarily large.

During the first two years of the war, the eye service of the American Ambulance treated about 500 cases. These were of all degrees of severity and consisted of traumatic conjunctivitis, foreign bodies (intra- and extra-ocular), traumatic cataracts, tears of choroid and retina and detached retina, injuries to optic and oculomotor nerves, wounds of orbit causing loss of lids and conjunctival sacs and in some cases of the bony parts of the orbit, cerebral injuries producing lesions of the optic centers and pathways.

Most enucleations were done under *local injections* of novocain and adrenalin. This was due mainly to the fact that the administration of ether would have exposed the patients to pneumonia, because of the accompanying face injuries, which were usually

present. The percentage of blind in the American Ambulance was 3 per cent.

The projectiles causing the ocular wounds were as follows in their order of frequency: (1) exploding shells, (2) rifle and machine-gun balls, (3) grenades, (4) bombs, (5) shrapnel. Their mode of action was:

(1) Contusion of the globe by either direct or indirect action.
(2) Rupture of the membranes with or without penetration.
(3) Infection of the globe after injury.
(4) Section or injury to the optic or oculomotor nerves.
(5) Intracranial lesions.

DECEMBER 21

Bilateral Choked Disk following Thyroidectomy

Dr. Frederick Krauss presented the history of a patient, male, aged twenty-three years, who had intense perspiration, delirium, tetany, and dizziness after an operation for thyroidectomy. Two months later he was still intensely nervous, depressed with the almost constant attacks of blindness, though his central vision was practically normal. He had an abnormally large red field crossing the form field. He had bilateral choked disk, the left eye showing a very small hemorrhage in addition. The swelling extended particularly along the course of the bloodvessels, subsiding in about six weeks. Six months later there was no sign of the trouble, except very full lymphatics on a very gray disk. Vision and the nervous system had apparently returned to normal lines.

Dr. Krauss felt that the traumatism in the removal of this cystic goitre had injured the sympathetic nerves in this region, causing a severe vasomotor reaction which produced an edema of the brain and optic nerves, with extension to the retina. These disappeared, leaving no permanent results.

Aviator's Dazzling

Referring to the subject of aviator's dazzling, Dr. T. B. Holloway alluded to the recent large appropriations made by Congress for the use of the Aviation Service and the establishment of schools for aviation. He referred to a lieutenant in the navy who had come under his observation during the summer of 1915. While on duty at an aviation station his eyes became somewhat painful, slightly injected, and there was some burning sensation of the lids. This was accompanied by poor ocular endurance for near work. A competent oculist who saw him about this time reported that he had some ciliary spasm, retinal hyperemia, and marked irritation of the conjunctivæ of the eyeballs and lids. After the correction of a compound hyperopic astigmatism he was advised to discontinue his aviation work. From that time on his eyes showed some improvement, but there was no complete relief. Later on he was assigned to destroyer duty, and after a trial trip many of the old symptoms returned. At this time he came under Dr. Holloway's observation, and the only change appreciable was a hyperemia of the conjunctivæ. There were no fundus changes, and with a suitable correction 6/6 vision was obtained with each eye. He was forbidden work that would subject him to glare. He resumed his duties at the aviation station in the fall, and with the use of a tinted lens he reported that he was quite comfortable.

Dr. Holloway alluded to some of the literature relating to the effects of glare, and called attention to the recent articles of Zade, who has described numerous cases observed in the German army, not only in the aviation corps, but especially among the anti-air craft gun crews. This observer also reports the frequent occurrence of a peripheral scotoma.

In closing, Dr. Holloway stated that he was very sorry he had not made a perimetric examination of the case, but at the time the patient came under observation Zade's work had not been reviewed.

Dr. Holloway stated that he thought he had suggested amber-tinted glasses, although he had failed to note this on his records.

He thought that while Zade had recommended a euphos tint, probably one of a dozen different tints might answer the same purpose.

Lupus of the Lids and Cornea

Dr. L. W. Hughes, by invitation, presented a patient, male, aged twenty-two years, who at first complained of poor vision in the left eye and swelling of the lids. The family history was negative except that his mother, one brother, one sister, and an aunt died of tuberculosis. His father and one brother were living and well.

The patient had always been well except for an occasional headache. Denied ever having had any venereal infection. He used tobacco and alcohol moderately.

In August, 1914, the patient first noticed what he called a "small pimple" on the lower lid of the left eye near the outer canthus. This rubbed his eye each time he closed his lids He received treatment from his family physician and the pimple disappeared. About three or four weeks ago, while at work, some powdered slaked lime got into his eyes. Both eyes became red, swollen and sore and upon looking at the eye he first noticed a little red mass beneath the upper lid about twice the size of a pinhead. It became larger but did not cause him any inconvenience.

The eye began to discharge about the latter part of September of the same year. He was then sent to the Philadelphia General Hospital and the condition was diagnosed as a tuberculoma of the eyelid. This growth was removed by Dr. Hansell on October 4, 1914, and the laboratory report by Dr. R. C. Rosenberger proved the former diagnosis to be correct.

At the time of the report the examination of O. S. showed the upper lid to be swollen and the margin of the lid appeared to be roughened. Upon eversion of the lid there were a number of soft dark red papules that gave the typical "apple-jelly" nodular appearance first described by Mr. Jonathan Hutchinson. At the centre of the lid was a furrow or sulcus as an evidence of where the

old tuberculoma was removed. There was swelling and hyperemia of the conjunctiva and some drooping of the eyelid. A corneal ulcer was seen at the left lower quadrant, with considerable bulging of the cornea. Vision 6/30.

There was a number of enlarged glands of the neck which had broken down and a discharging sinus on the left side of the face near the ear.

Dr. Hughes summarized the case as follows:

1. Family history of tuberculosis.

2. Involvement of the lids and cornea was independent of the involvement of the nose and cervical glands.

3. Removal of the tuberculoma and its positive diagnosis by laboratory reports.

4. Slow growth.

5. Repeated negative Wassermann ruled out syphilis.

6. Improvement of general condition by good food, rest, fresh air, and light exercise.

Dr. C. E. G. Shannon stated that at the time of the patient's admission to the hospital the upper lid was swollen, and on eversion of the lid a growth about the size of a small walnut was seen. It was polypoid in form and granular in appearance, and was attached to the tarsal conjunctiva by a short, thick pedicle. The surrounding conjunctiva was occupied by a great number of papillary growths, while the surface of the lid was bathed with a mucopurulent secretion. The present appearance of the conjunctival lesion shows a marked excavation at the site of the former growth surrounded by a preponderance of papillary proliferations.

Dr. Shannon said that involvement of the cornea as shown in this case is rather frequent in lupus of the conjunctiva, and therefore we should always regard the prognosis as grave, as extension of the process may cause destruction of the cornea or even the eyeball itself.

Burton Chance, in commenting upon Dr. Hughes's case of lupus of the eyelid, gave an outline in both the dermatological and the ocular aspects of this manifestation of tuberculosis, a

disease which occurs so rarely in the practice of ophthalmologists. He stated that he had not had a patient exhibiting active symptoms but he had had several who had applied for relief from the effects of the disease of the lids, the ducts, and of the globes. In these there were quite severe erosions of the skin, notably of the alæ of the nose, in which region it is so stated by dermatologists that it frequently first shows itself, and is well shown in the case before us. Chance's cases had been in blondes and mostly Irish-born; he does not recall a case in a negro.

Lupus is so rare that few ophthalmologists, and dermatologists too, have seen it in America, yet it is comparatively common in European clinics.

The disease is a chronic cellular infiltration of the skin, producing nodules beneath the surface, which increase in size and form patches. These patches may become absorbed or may ulcerate, to be followed by cicatrization. The origin of the disease depends upon the invasion of the tubercle bacillus. It is manifested by a combination of proliferation and ulceration, granulation and cicatrization, affecting chiefly the corium. The nodules consist of cells grouped around the capillaries and contain giant cells, yet the tubercle bacilli, although pathognomonic, are few in number and are not always present in all specimens, although they were found in the nodules excised from the lid in the present case. The reactions to tuberculin tests notwithstanding are completely positive. The mode of invasion is uncertain; it may be by direct inoculation; by extension from deeper foci; through the circulation; and even by maternal transference.

The patients usually present the scrofulous cachexia, with more or less adenitis, but, except for the ulceration, they are remarkably free from subjective symptoms.

The mucous membranes may be invaded, and, when the disease has lasted a long time, the eyelids and the globe may become involved. The lids may become distorted, the conjunctiva contracted, and the cornea destroyed in the serious cases. The disease lasts many years, is rebellious to treatment, but in America the course and symptoms are less protracted and severe than in Europe.

The treatment consists in the pursuance of such general regimen as should be applied in cases of a more general tuberculosis and by immunization by tuberculin inoculations. Locally, pastes may be used; radiant emanations; Finsen light; high-frequency currents in the manner of desiccation as being among the most efficient methods. When it invades the eye the ulceration should be excised or curetted and the raw surfaces cauterized. Not much hope can be held out when it invades the globe, especially the cornea.

Dr. Chance said that he had been so much impressed by the beneficial results obtained from the Maxwell operation in certain cases under Dr. Zentmayer's care that he himself had performed it two or three years ago in a case, of which he had endeavored to have the patient present. In that case the lower lid had been drawn far into the orbit, and the upper lid was a mere nodule. By the Maxwell operation the patient had been pleased to wear a modified glass eye, and three weeks ago submitted to operation to restore the position of the upper lid by means of the method described by Dr. Schwenk before this Section in December, 1915. The immediate effects of the last operation brought about the formation of a distinct cul-de-sac, which, however, has contracted somewhat in the past few days. Because of the benefit derived from Maxwell's procedure on the upper lid in Dr. Zentmayer's patient, Chance is considering to attempt it in his case, should the contraction become greater. The Maxwell operation not only increases the capacity of the orbital socket, but also succeeds admirably in the restoration of the contour and function of the eyelids.

Implantation Cyst of the Anterior Chamber

Dr. Harold G. Goldberg presented a case of implantation cyst of the anterior chamber following a punctured wound of the eyeball received several months before the appearance of the growth. The cyst was of the pearl tumor type composed of both epithelium and connective tissue which had been conveyed to the anterior

chamber through the original wound. Two attempts were made to remove it by operation, each time including a section of the iris. The vision of the eye was very little affected, and there was no evidence of any extension into the deeper structures. Dr. Goldberg believed, however, that the iris had become so deeply invaded by the epithelial growth that a thorough removal was impossible.

Double Maxwell Operation

Dr. William Zentmayer exhibited a case of contracted socket in which a double Maxwell operation had been performed five years previously. The pedicle grafts in the cul-de-sac had not shrunken in the least and the patient was wearing an artificial eye with comfort. The levator which had to be cut in order to slip the skin graft through the incision made through the thickness of the lid had its function unimpared.

Hydrophthalmos

Dr. Zentmayer showed an extreme case of hydrophthalmos. The right eye was blind and soft. The left eye had vision = hand movements, tension stony, hard. Dr. Zentmayer discussed the hypotension of the right eye in the light of Treacher Collins's studies regarding the phenomenon in contusions of the globe, and concluded that it was probably the result of the rupture of the sclera from striking the eye, as the child's forehead bore many scars received in this way.

J. Milton Griscom, M.D.,
Clerk.

REPORT OF THE SECTION ON OTOLOGY AND LARYNGOLOGY

In accordance with the By-laws of the College, the Clerk of the Section respectfully submits the following report:

The membership of the Section on November 30, 1916, was 26 as compared to 27 last year.

The total attendance at the six meetings was 171; the average attendance was therefore 28. The average attendance of members only was 11.

Fourteen subjects were presented and discussed.

The officers of the Section elected December 15, 1915, are:

Chairman—Dr. George C. Stout.

Clerk—Dr. Benjamin D. Parish.

Executive Committee—Dr. G. Morley Marshall, Dr. Ralph Butler, Dr. Benjamin D. Parish.

List of Papers

December 15, 1915

Dr. George Fetterolf: "Galvanocauterization as a Substitute for Tonsillectomy in certain Selected Cases."

Dr. J. Clarence Keeler (by invitation): (1) "Report of a Case of Bilateral Mastoiditis in a Child; Extradural Abscess Complicated by Pneumonia; Operation and Recovery. (2) Report of a Case of Chronic Otorrhea in a Child, followed by Mastoiditis and Facial Paralysis, Pericardial Effusion, and Tubercular Adenitis; Operation and Recovery."

January 19, 1916

Dr. D. Braden Kyle: "Report of the Removal of a Large Rhinolith." Exhibition of specimen.

Dr. Alfred Gordon (by invitation): "Septic Intracranial Thrombosis Accompanied by Sinusitis and Terminating in Brain Abscess." Presentation of brain.

Dr. C. C. Eves: "Perichondritis of the Larynx Complicating Typhoid Fever."

February 16, 1916

Dr. B. Alexander Randall: "A Note on a Case of Hysterical Deafness."

Dr. Fielding O. Lewis: "Report of a Case of Lupus of the Nose."

Dr. John C. Stimson (by invitation): "What are the After-results of Tonsillectomy? A Study of 571 Cases."

March 15, 1916

Dr. Samuel J. Kopetzky (by invitation): "The Significance of Laboratory Findings for Diagnosis and Treatment in Otology." Discussed by Dr. Randall, Dr. Smith, and Dr. Kolmer.

April 19, 1916

Dr. George Fetterolf: (1) "Presentation of a New Internal Speculum for Submucous Resection of the Nasal Septum. (2) Report of a Unique Case of Papilloma of the Uvula." Presentation of Specimen.

Dr. Ralph Butler: "An Unusual Case of Thrombosis of the Lateral Sinus." Presentation of patient.

Dr. James A. Babbitt: "Some Observations on Radium Therapy in the Nose and Throat."

Dr. C. C. Eves: "A Preliminary Report in a Series of Ten Cases of the Removal of Foreign Bodies from the Bronchi, Trachea, and Esophagus by the Direct Method."

November 15, 1916

Dr. Edgar M. Holmes (by invitation): "Rhinoscopy; A Review of the Use of the Periscope in Diagnosis and as an Aid in Treatment." Discussed by Dr. Fetterolf, Dr. Butler, and Dr. Lewis.

Benjamin D. Parish,
Clerk.

REPORT OF THE SECTION ON GENERAL MEDICINE

The Clerk of the Section respectfully submits the following report in accordance with the By-laws of the College:

The Section has had a prosperous year under the leadership of Dr. James E. Talley, elected Chairman, January 26, 1914, and Dr. Clifford B. Farr, elected Chairman, January 24, 1916. The Executive Committee of the Section appointed by the President of the College for the year 1916 were Dr. M. Howard Fussell, Dr. Clifford B. Farr, and Dr. James E. Talley.

The Section has an active membership of 78 Fellows. During the year eight regular meetings were held, at which 27 papers were read and two patients exhibited. Seventy-six members or guests discussed these twenty-seven communications.

The total attendance at the eight meetings totaled 328, and of this number 123 were members of the Section.

The average attendance at the meetings was 41 and the average number of Section members who attended was 16.

On April 20, 1916, Dr. W. B. Cannon, of Harvard University, read a paper before the Section entitled "An Explanation of Some Disorders Supposed to have an Emotional Origin."

In connection with a paper read by Dr. R. Max Goepp and Dr. John A. Kolmer (by invitation), on May 22, 1916, entitled "Trichiniasis," there was exhibited through the courtesy and thoughtfulness of Dr. James Tyson, slides showing the trichina in the tissues that had been mounted by Dr. Tyson forty-seven years before.

The following is a complete list of the communications read before the Section during the past year.

List of Papers

December, 1915

Exhibition of a Patient Showing Many Evidences of Hypopituitarism. By Dr. N. L. Boston.

Lead Encephalopathy, with Remarks upon Treatment. By Dr. G. V. Emmerson.

An Analysis of 147 Cases of Drug Addiction at the Philadelphia General Hospital. By Dr. George E. Price and Dr. Joseph McIver (by invitation).

Erythema Nodosum and Pulmonary Tuberculosis. By Dr. Charles W. Luders.

A Case of Sarcoma of the Stomach Discovered at Operation. By Dr. T. G. Schnabel.

A Case of Sarcoma of the Stomach, with Autopsy Findings. By Dr. C. B. Farr.

January, 1916

Influenzal Meningitis, with Report of a Case. By Dr. Robert G. Torrey.

Bronchiectasis and its Treatment, with Special Reference to Vaccine Treatment. By Dr. Alfred Stengel and Dr. Herbert Fox.

Significance of Functional Renal Tests in Prognosis. By Dr. Joseph Sailer.

February, 1916

Causes and Treatment of Hemorrhage in Pulmonary Tuberculosis. By Dr. Joseph Walsh.

The Treatment of Sciatica. By Dr. Alfred Gordon.

A Consideration of Cardiospasm. By Dr. B. B. Vincent Lyon.

Delayed Gastric Digestion. By Dr. Martin E. Rehfus.

March, 1916

Spelter Chills. By Dr. David Riesman and Dr. Russell S. Boles (by invitation).

The Causation of Insanity. By Dr. Charles Burr.

The Determination of Intrapleural Pressure. By Dr. Charles M. Montgomery.

A Consideration of the Naegleschmidt-Bergonie Method of Treating Obesity and Metabolic Disturbances. By Dr. B. B. Vincent Lyon.

April, 1916

An Explanation of Some Disorders Supposed to Have an Emotional Origin. By Dr. W. B. Cannon.

May, 1916

An Unusual Type of Cardiac Arrhythmia Relieved by Surgical Operation. By Dr. E. B. Krumbhaar.

Trichiniasis: A Clinical Report, with Demonstration of the Parasites in the Tissues. By Dr. R. Max Goepp and Dr. John A. Kolmer (by invitation).

(In connection with this paper Dr. Goepp exhibited slides showing the trichina in the tissues that had been mounted by Dr. James Tyson forty-seven years ago.)

A Brief Report of Several Papers Read before the Association of American Physicians in Washington. By Dr. H. R. M. Landis.

A Brief Report of Several Papers Read before the Association of American Physicians. By Dr. Joseph Sailer.

October, 1916

Surgical Experiences, with Encapsulated Empyema and Abscess of the Lung; a Plea for Exploratory Thoracotomy (Lantern Slides). By Dr. Astley P. C. Ashhurst.

The Intragastric Diagnosis of Ulcer of the Stomach. By Dr. Martin E. Rehfus (by invitation).

Metabolic Studies upon a Case of Angioneurotic Edema. By Dr. O. H. Perry Pepper and Dr. T. Grier Miller (by invitation).

November, 1916

Roentgen Studies of the Action of Pituitrin upon the Gastro-intestinal Tract of Man. By Dr. Henry K. Pancoast and Dr. Arthur H. Hopkins.

The Treatment of Cases of Chronic Gastritis. By Dr. B. B. Vincent Lyon.

A Report of Certain Unusual Cases of Malaria, with a Brief Analysis of Fifty Cases of this Disease. By O. H. Perry Pepper.

E. J. G. Beardsley,
Clerk.

LIST OF PRIZES AND LECTURES

THE WILLIAM F. JENKS MEMORIAL PRIZE

(Triennial)

1889	John Strahan, M.D.	Belfast (Ireland).
1895	Abram Brothers, M.D.	New York.

Note—June 12, 1900, the William F. Jenks Memorial Prize Fund was transferred to the Library as the "William F. Jenks Memorial Library Fund."

ALVARENGA PRIZE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

(Annual)

1890	R. W. Philip, M.D.	Edinburgh (Scotland).
1891	L. Duncan Bulkley, M.D.	New York.
1892	R. H. L. Bibb, M.D.	Saltillo.
1894	G. E. de Schweinitz, M.D.	Philadelphia.
1895	Guy Hinsdale, M.D.	Philadelphia.
1897	Joseph Collins, M.D.	New York.
1898	S. A. Knopf, M.D.	New York.
1899	Robert Randolph, M.D.	Baltimore.
1900	David de Beck, M.D.	Cincinnati.
1901	George W. Crile, M.D.	Cleveland.
1903	William S. Carter, M.D.	Galveston.
1905	D. Chalmers Watson, M.D.	Edinburgh (Scotland).
1907	William Louis Chapman, M.D.	Providence.
1908	William T. Shoemaker, M.D.	Philadelphia.
1910	M. Katzenstein, M.D.	Berlin (Germany).
1911	Francis D. Patterson, M.D.	Philadelphia.
1914	H. B. Sheffield, M.D.	New York.
1915	J. E. Sweet, M.D.	Philadelphia.

NATHAN LEWIS HATFIELD PRIZE FOR ORIGINAL RESEARCH IN MEDICINE

(Triennial)

1901	Henry F. Harris, M.D.	Atlanta.
1909	Martin Henry Fischer, M.D.	Oakland.

NOTE.—November 29, 1913, by Supplemental Deed of Trust, the title of this Fund was changed to "Nathan Lewis Hatfield Prize and Lectureship."

WEIR MITCHELL LECTURES

Jan. 17, 1911	Arthur R. Cushny, M.D.	London.
Mar. 30, 1911	Edmund B. Wilson, Ph.D., LL.D.	New York.
May 16, 1911	Svante Arrhenius	Stockholm.
Nov. 3, 1911	William T. Porter, M.D.	Boston.
Mar. 29, 1912	William H. Howell, M.D.	Baltimore.
Oct. 21, 1912	G. H. F. Nuttall, F.R.S., M.D.	Cambridge (England).
April 4, 1913	H. P. Armsby, Ph.D., LL.D.	Pennsylvania.
Feb. 25, 1914	Harvey Cushing, M.D.	Boston.

PUBLIC LECTURES

Feb. 16, 1910	S. Weir Mitchell, M.D.	Philadelphia.
Nov. 17, 1910	Simon Flexner, M.D.	New York.
Dec. 15, 1910	William H. Welch, M.D.	Baltimore.
April 18, 1911	James G. Mumford, M.D.	Clifton Springs, N. Y.
Nov. 20, 1911	Talcott Williams, A.M., LL.D., Litt.D.	Philadelphia.
April 29, 1912	Owen Wister, A.M., LL.D.	Philadelphia.
Feb. 17, 1913	John K. Mitchell, M.D.	Philadelphia.
Feb. 15, 1916	Daniel J. McCarthy, M.D. and Walter Estell Lee, M.D.	Philadelphia.
April 15, 1916	Surgeon A. M. Fauntleroy	U. S. Navy.

MÜTTER LECTURES

1865	J. H. Packard, M.D.	Philadelphia.
1866	J. H. Packard, M.D.	Philadelphia.
1867	J. H. Packard, M.D.	Philadelphia.
1868	Harrison Allen, M.D.	Philadelphia.
1869	J. H. Brinton, M.D.	Philadelphia.
1872	J. da S. Solis Cohen, M.D.	Philadelphia.
1879	S. W. Gross, M.D.	Philadelphia.
1882	E. O. Shakespeare, M.D.	Philadelphia.
1885	H. F. Formad, M.D.	Philadelphia.
1888	O. H. Allis, M.D.	Philadelphia.
1890–1891	Roswell Park, M.D.	Buffalo.
1893–1894	De Forrest Willard, M.D., and Guy Hinsdale, M.D.	Philadelphia.
1896	O. H. Allis, M.D.	Philadelphia.
1899–1900	J. B. Roberts, M.D.	Philadelphia.
1901	H. W. Cushing, M.D.	Boston.
1902	L. A. La Garde, M.D.	Washington.
1903	C. N. B. Camac, M.D.	New York.
1904	G. H. Monks, M.D.	Boston.
1905	A. O. J. Kelly, M.D.	Philadelphia.
1906	W. J. Mayo, M.D.	Rochester, Minn.
1907	J. Rogers, M.D., and S. P. Beebe, M.D.	New York.
1908	G. W. Crile, M.D.	Cleveland.
1909	H. D. Fry, M.D.	Washington.
1910	T. W. Hastings, M.D.	New York.
1911	C. F. Nassau, M.D.	Philadelphia.
1912	J. C. Bloodgood, M.D.	Baltimore.
1913	R. C. Coffey, M.D.	Portland, Ore.
1914	F. H. Albee, M.D.	New York.
1915–1916	Rudolph Matas, M.D.	New Orleans, La.
1916	Nelson M. Percy, M.D.	Chicago, Ill.

ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1916

MR. PRESIDENT: In accordance with the ordinances and By-Laws of the College, I herewith submit the following report of the Library Committee for the year 1916:

Total number of volumes in the Library, including the bound volumes and 15,367 unbound "Reports" and "Transactions"	115,346
Number of unbound "Theses" and "Dissertations" .	8,577
Number of unbound pamphlets	92,789

Included in the above total there are 3,479 volumes known as "reserves," consisting of second copies of some of the more important periodical publications, and 2,993 volumes more or less incomplete.

The duplicates, which are not included in the above total, number 4,568.

The following table shows the number of volumes in the various divisions of the Library:

	Bound.	Incomplete and unbound.	Total.
General Library . . .	79,614	2,946	82,560
Lewis Library	13,596	44	13,640
On permanent deposit:			
S. D. Gross Library . .	3,559	3	3,562
Library of the Obstetrical Society of Philadelphia .	217	....	217
			99,979

Received during the year from all sources 3,936 volumes, 13,070 pamphlets and 18,608 numbers of various periodicals.

Divided as follows:

	Volumes.	Pamphlets.	Journals.
General Library	2,910	12,918	18,608
Lewis Library	10		
S. D. Gross Library	5		
By purchase from General Fund	418		
In exchange	595	152	
	3,938	13,070	18,608

Accessions (including 25 volumes "reserves"):	
General Library	2,436
Lewis Library	10
S. D. Gross Library	5
	2,451

Total increase in number of volumes for the year, 2,451.

Photographs received in response to requests sent out during the year:

Fellows of the College, 29. Foreign, 1.
Total number of portraits listed, 7,577.

The individual "donors" for the year ending November 1, 1916, number 443; this represents 892 distinct presentations. Each gift is duly acknowledged and properly recorded.

The following list shows the donations of 25 volumes or more, and the number of volumes presented by the various publishing houses:

	Volumes.
Dr. S. M. McCollin	171
Dr. R. S. Hooker	122
Dr. F. P. Henry	113
Dr. F. D. Patterson	95
Dr. G. M. Piersol	77
Dr. C. K. Mills	74
Dr. W. H. Bennett	54
Dr. Samuel G. Dixon	48
Dr. Francis R. Packard	47
Dr. Joseph Sailer	47
Dr. B. D. Parish	46
Dr. Oscar H. Allis	40
Dr. C. W. Burr	39
Mrs. C. R. Heed	39
Dr. W. G. Spiller	32
Dr. H. A. Hare	29
Dr. W. W. Keen	27
Dr. J. C. Wilson	27
Dr. David Riesman	25

From the publishing houses of:

D. Appleton & Company	1
P. Blakiston's Son & Co.	19
Ch. Boulangé, Paris	11
F. A. Davis Company	14
Lea & Febiger	1
J. B. Lippincott Company	14
Salvat y Compañia	2
W. B. Saunders Company	48
William Wood & Company	5

The Library is indebted for large gifts of pamphlets and unbound periodicals to the following donors:

Dr. Mary C. Allen
Dr. Oscar H. Allis
Dr. Charles Baum
Messrs. P. Blakiston's Son & Co.
Dr. Burton Chance
Dr. John M. Cruice
Dr. Edward P. Davis
Dr. G. G. Davis
Dr. F. X. Dercum
Dr. C. W. Dulles
Dr. A. A. Eshner
Dr. Charles H. Frazier
Dr. E. H. Goodman
Dr. J. P. Crozer Griffith
Dr. S. McC. Hamill
Dr. Hobart A. Hare
Mrs. C. R. Heed
Dr. F. P. Henry
Dr. A. Bern Hirsh
Dr. R. S. Hooker
Dr. William W. Keen
Dr. John A. Kolmer
Dr. Robert G. LeConte
Dr. Morris J. Lewis
Messrs. J. B. Lippincott Company
Dr. G. Morley Marshall
Dr. Edward Martin
Dr. Wm. J. Merrill
Dr. Charles K. Mills
Dr. John K. Mitchell
Dr. Elliston J. Morris
Dr. George W. Norris
Dr. G. W. Outerbridge
Dr. F. R. Packard
Messrs. W. B. Saunders Company
Dr. A. C. Sautter
Dr. George E. de Schweinitz
Dr. William G. Spiller
Dr. L. W. Steinbach
Dr. H. W. Stelwagon
Dr. C. F. Taylor
Dr. T. Turner Thomas
Dr. Joseph P. Tunis
Dr. Henry R. Wharton
Dr. J. William White
Dr. James C. Wilson
Dr. C. S. Witherstine
Dr. William Zentmayer
Dr. M. W. Zimmerman

734 new publications were added to the Library during the past year. 41 of these were written or edited by Fellows of the College.

30 volumes were presented by the following authors or editors:

Dr. Astley P. C. Ashhurst (editor)
Dr. Francis G. Benedict
Mrs. A. A. Bliss (editor)
Sir T. Lauder Brunton, Bart.
Dr. Francis X. Dercum
Mr. H. H. Donaldson (compiler)
Dr. Thomas Addis Emmet
Dr. R. H. Ferguson (compiler)
Dr. Herbert Fox
Dr. Hobart A. Hare (editor)
Dr. F. L. Hoffmann
Dr. C. W. Hollopeter
Dr. Edward Jackson (editor)
Dr. J. Köräsi
Dr. Stuart McGuire
Dr. G. W. Mackenzie
Dr. George W. Norris
Dr. G. Delgado Palacios
Dr. John B. Roberts
Dr. E. A. Schumann (editor)
Dr. P. G. Skillern, Jr.
Dr. J. E. Sweet
Mr. Edward Trust (compiler)

18 volumes were sent by the publishers at the request of the following authors or editors:

Dr. James M. Anders
Dr. P. Andreae
Dr. A. Max Goepp
Dr. George M. Gould
Dr. E. E. Graham
Dr. William W. Keen
Dr. John A. Kolmer
Dr. H. R. M. Landis (editor)
Dr. Joseph McFarland
Dr. C. E. de M. Sajous
Dr. George E. de Schweinitz
Dr. Louis Starr
Dr. James Thorington
Dr. W. S. Wadsworth

Summary of the Funds:

	Volumes purchased.	Cost.
Henrietta Rush Fales Baker Fund	61	$98.05
Luther S. Bent Fund	8	12.97
William T. Carter Fund	32	91.44
Girardus Clarkson Fund	2	2.08
Francis X. Dercum Fund	46	135.54
Louis A. Duhring Fund	15	36.96
John D. Griscom Fund	18	83.18
William F. Jenks Fund	42	152.41
Oliver A. Judson Fund	..	....
William V. and John M. Keating Fund	18	44.77
William W. Keen Fund	20	78.21
Library Endowment Fund	43	187.20
Horace Magee Memorial Fund	207	572.47
J. Ewing Mears Fund	24	78.62
Charles K. Mills Fund	5	7.65
Weir Mitchell Fund	13	52.85
John H. Musser Fund	9	27.59
Elizabeth K. Newcomet Fund	14	27.34
William F. Norris Fund	28	171.28
Charles A. Oliver Fund	2	4.22
Philadelphia Medical Society Fund	2	5.47
Lewis Rodman Fund	7	45.18
Douglas Stockton Warren Fund	24	49.05
John F. Weightman Fund	2	10.81
Caspar Wistar Fund	49	137.58
	691	$2,112.92

Special Accounts:

	Volumes purchased.	Cost.
Fund for completing files of journals	200	$524.99
Fund for rare and valuable books	20	1,257.66
New Book Fund	17	30.69
S. D. Gross Library Account	3	4.80
	240	$1,818.14

George B. Wood Fund for Library supplies, etc. Expended $308.47.

Morris Longstreth Fund. Expended on account of Salaries, $1,156.89.

Catalogue Endowment Fund. Expended on account of Salaries, $40.35.

	1916.	1915.	Increase.
Books bound	1,253	953	300

	1916.	1915.	Decrease.
Number of Visitors to the Library	5,910	8,270	2,360
[Fellows of the College:	1,864	3,263	1,399]

The Library has been kept open two evenings each week and on the six minor legal holidays, for the same hours and period of time, as during the preceding year.

	1916 (77 evenings).	1915 (75 evenings).	Decrease.
Visitors, evening	553	690	137
[Fellows of the College	156	186	30]
Visitors, legal holidays	74	125	51
[Fellows of the College	17	24	7]

The above figures are included in the total number of visitors for the year.

	1916.	1915.	Decrease.
Number of books consulted in the Library	18,808	25,770	6,962

The number of books reported as "consulted in the Library" includes only those supplied on demand. Readers have access

to the bound volumes kept in the Reading-room, and the Fellows of the College have access to the Book-stacks. There are therefore a great many volumes consulted of which no accurate record can be kept.

	1916.	1915.	Decrease.
Number of books taken out . .	4,533	4,799	266
	Works.	Volumes.	Cards written.
Cataloguing	993	1,206	12,030

All the books added to the shelves during the past year, and 323 of the more important pamphlets have been catalogued, and each volume has been accessioned and shelf-listed.

12,791 unbound pamphlets and reprints have been subject-headed and arranged alphabetically by subject, and by author under the subject. The work of making this important change in the arrangement of the unbound pamphlets and reprints is now about completed, and the binding by subjects will be commenced during the coming year.

Revision of catalogue: Number of cards revised, typewritten, examined and filed for the year ending November 1, 1916: 15,911.

The revision of the catalogue has been completed as far as the subject "Veratrine" in the second series of the "Index-Catalogue."

The current periodical publications, including "Transactions" and "Reports," received as issued, at this time are obtained through the following sources:

	American.	Foreign.
Endowment Funds	17	292
By purchase from General Account . . .	45	415
In exchange	95	91
Editors	124	21
Publishers	57	2
In addition, current numbers of periodicals are received, at stated intervals, through the courtesy of the editors and editorial staff of the following journals: American Journal of the Medical Sciences Therapeutic Gazette	145	44
	483	865

Actual number of current periodicals received at this date, 943.

New subscriptions added during the past year: American 15; foreign, 66. Total, 81.

The following is a list of the foreign schools of medicine with which we exchange publications:

University of	Amsterdam	University of	Königsberg
"	Basel	"	Lausanne
"	Berlin	"	Leiden
"	Bern	"	Leipzig
"	Bonn	"	Liège
"	Breslau	"	Lund
"	Erlangen	"	Marburg
"	Geneva	"	Rostock
"	Giessen	"	Strassburg
"	Göttingen	"	Upsala
"	Greifswald	"	Utrecht
"	Halle	"	Würzburg
"	Heidelberg	"	Zurich
"	Kiel		
Faculty of	Medicine of	Bruxelles	
"	"	Paris	
"	"	Toulouse	
"	"	Yucatan	

The above list has been allowed to stand as it was prior to the war in Europe; while it is hoped that after the disturbance has ended all the institutions named will resume their duties, yet it seems quite possible that a revision will have to be made. During the past year we have heard from Amsterdam, Bern, Giessen, Göttingen, Lausanne, Leipzig and Toulouse.

The Bureau of International Exchanges will only deliver parcels to certain designated places in Europe; and, in consequence, the *Transactions* of the College for a number of our foreign exhanges must be held in waiting.

378 dissertations have been received, and of these 352 were added to our collection during the year.

We have received for the current year ending November 1, 1916, in cash, from the sale of duplicates, $281.68.

We have an exchange, arranged on a cash basis, with two New York dealers, and have received during the past year books and journals valued at $48.35; and have a balance to our credit of $116.46.

During the year we have distributed duplicate books and journals on exchange account to the following:

Boston Medical Library
Hygienic Laboratory, United States Treasury Department
Kansas City Medical Library Association
Ramsay County Medical Society Library
United States Department of Labor
University of Texas, Medical Department

Returns have been received from Boston Medical Library, Hygienic Laboratory, United States Treasury Department, Kansas City Medical Library Association, McGill Medical Library, Medical Library Association, Ramsay County Medical Society Library, University of Michigan Library, and University of Texas Medical Department.

With the aid of exchanges, and by purchase from funds appropriated for the purpose by the Library Committee, we have, since November 1, 1915, completed our files of the following journals:

American Labor Legislation Review, New York.
Archiv für Protistenkunde, Jena.
Archives Italiennes de Biologie, Turin and Pisa.
Bulletin de Laryngologie, Otologie et Rhinologie, Paris.
Chemical Abstracts, Easton, Pa.
La Chronique Médicale, Paris.
L'Experience, Paris.
Internationales Centralblatt für Ohrenheilkunde, Leipzig.
Journal of Race Development, Cambridge, Mass.
Revue Médico-Chirurgicale des Maladies des Femmes, Paris.

Amount of fines collected from November 1, 1915, to November 1, 1916, $34.75.

The following is a list of the rare medical books and works of special interest received during the past year:

Incunabula

(Total number of incunabula at this date, 193)

Abiosus, Joannes. Dialogus in astrologiæ defensionum. Venice, Franciscus Lapicida, 1494. [Hain 24.]

Very rare work, and the only work printed by Lapicida. This copy was bound by Rivière.

Fund for Rare Books.

Albucasis. Incipit liber servitoris. [Simple medical preparations.] Venetiis, Nicolaus Jenson, 1471. [Copinger 3450.]

Only a few copies known. Very valuable from a typographical point of view, being a dated Jenson work of 1471.

Fund for Rare Books.

Alchabitius. [Libellus isagogicus de planetarum conjunctionibus.] Venetiis, Johannes & Gregorius de Gregoriis, 1491. [Hain 618.]

An unusually interesting copy. The ordinary edition is described by Hain 618. This copy, however, has a number of pages printed in a type larger than that used in the bulk of the work; and the first line of b 3 belongs to some other medical book.

Fund for Rare Books.

Benedictus, A. De observatione in pestilentia. Venetiis, per Johannem et Gregorium de Gregoriis, 1493. [Hain 807.]

Fund for Rare Books.

Censorinus. [De die natali.] Bononiæ, Benedictus Hectoris, 1497. [Hain 4847.]

Fund for Rare Books.

de Thienis, Gaietanus. [Recollectæ super viii libb. physicorum Aristotelis.] [Tarvisium, Hessen, 1474.] [Hain 15496.]

Important and little-known work by the third printer in Treviso, who printed only two works.

Fund for Rare Books.

Works of Special Interest

Aristotelis. Utilissimus liber. . . de secretis secretorum. Burgos, Andrea de Burgos, 1505.

Black letter. Only a few copies known.

Fund for Rare Books.

Arnoldus de Villa Nova [et al]. Le tresor des pauvres. Lyon, Nourry, 1518.

Fund for Rare Books.

Avicenna. Omnes canones prima fen quarti canonis Avicene. Papie, de Garaldis, 1517.

Five parts in one volume. Extremely rare; unknown to most bibliographers.

Fund for Rare Books.

Browne, Sir Thomas. Pseudodoxia epidemica. Second edition. London, Miller, 1650.

Presented by J. Percy Keating, Esq.

B[ulwer], J[ohn]. Anthropometamorphosis. Man transform'd. London, Hunt, 1653.

Has many curious woodcuts. Bound by Rivière.

Fund for Rare Books.

Chappusius, Nicolaus. De mente et memoria libellus utilissimus. [Paris, Badius, 1515.]

Badius, the printer of this work, was the rival of Aldus in the issuing of classical publications.

Fund for Rare Books.

Ferrerius, Augerius. De pudendagra, lue hispanica libri duo. Antverpiae, apud M. Nutii viduam, 1564.

Ferrerius was physician to Catherine de Medici.

Fund for Rare Books.

Fuchs, Leonhard. New Kreüterbuch. Basell, Isingrin, 1543.

Very rare first German edition. This edition and the Latin edition of 1542 were regarded by William Morris as the finest illustrated Herbals ever produced.

Fund for Rare Books.

Ganivetus, Joh. Epistola astrologie defensiva. Lugdunenis, Kleyn, 1508.

A rare work.

Fund for Rare Books.

Hegendorff, Christopher. Encomium ebrietatis. Lipsie, Schumann, 1519.

Very rare edition.

Fund for Rare Books.

de La Vauguion. Compleat body of chirurgical operations. London, Bonwick, [1699].

Plates lacking.

Presented by Dr. M. W. Zimmerman.

London's dreadful visitation; or, A collection of all the bills of mortality for this present year. . . beginning with 20 December, 1664. London, Cotes, 1665.

Presented by Sir William Osler, Bart.

Mousin, J. Discours de l'yvresse et yvrongnerie. Toul., Philippe, 1612.

First edition from a rare Provincial press.

Fund for Rare Books.

Nicolaus Salicetus. Liber meditationum. Venetu, Lucantonius de Giunta Florentinus, 1501.

Fund for Rare Books.

Scot, Reginald. Discoverie of witchcraft. London, Brome, 1584.

Black letter. This copy is complete, containing the very rare pages of illustrations between pp. 352 and 353.

Fund for Rare Books.

Shuttleworth, John. Treatise of opticks direct. London, Midwinter, 1709.

Fund for Rare Books.

Taisnerius, Joannes. Opus mathematicum octo libros complectens. Coloniae Agrippinae, Baumius, 1583.

Fund for Rare Books.

Valles Covarrubianus, Franciscus. Controversiarum medicarum et philosophicarum. Lugduni, Rouillii, 1591.

By purchase.

Manuscripts

[Smith, Daniel B.] Notes on the lectures on midwifery in the University of Pennsylvania. [Philadelphia, 1810.]

Presented by the Library of Haverford College.

[Wood, George B.] Catalogue of pupils attending the lectures on materia medica and pharmacy at the University of Pennsylvania. [Philadelphia, 1840–1849.]

Presented by Miss Juliana Wood.

Other Interesting Additions

Königl. Friedrich-Wilhelms-Universität Berlin. Three-mark piece struck in honor of the first centenary, 1910.

Presented by Dr. W. W. Keen.

Pilcher, Lewis Stephen. Bronze medal to commemorate fifty years of practice.

Presented by the Pilcher Centennial Committee.

Royal Army Medical Corps. Several souvenirs of the Royal Army Medical Corps.

Presented by Dr. W. W. Keen.

Siamese Twins. Scrap Book.

A volume from the library of the late George Dunn, containing a collection of illustrations in black, white and colors, of the Siamese Twins; together with bills, posters and descriptive matter issued at the time of their exhibition in England. [Deposited in the Mütter Museum.]

Presented by Sir William Osler, Bart.

We have also received during the year many interesting and more or less valuable autograph letters.

The figures in the present report, when compared with the previous year, show a decrease in the number of visitors (2,360); the number of visits made by Fellows (1,399); the number of books consulted (6,962); and the number of books taken out (266). The "Study-rooms," on the other hand, show a decided increase in the number of volumes called for and retained in these rooms. The average has been 203 volumes, an average increase of 28 over the past year.

No improvement can be reported in regard to the receipt of foreign medical literature; on the contrary, the continuance of the war, with the restrictions imposed from varying circumstances, has led to a practical stoppage of mail and freight delivery from certain countries. For instance, comparing the number of subscriptions to periodical publications on our cards with the actual

number received during the past year, there is a loss in the foreign periodicals, other than Great Britain and her colonies, of more than 300. No periodicals in the German language have been received since March, 1916. Our agents state that most of the journals are being published and that a copy of every issue is held for each name on the subscription list, and that all will be delivered as soon as the necessary arrangements have been completed. We are confident, however, that since the beginning of the year 1914 quite a number of journals, as well as other serial publications, in the French, Italian and German languages, have ceased publication. How many, and what they are, will not be known until peaceful conditions have been restored and time allowed for readjustment.

In regard to books, there has been a considerable increase in the number of new publications received (160). We stated in last year's report that when compared with 1913 there was a loss of about 90 per cent. of books published in the French language; about 69 per cent. of books published in the German language, and about 34 per cent. in the publications of Great Britain. Making the same comparison between the years 1913 and 1916, we find a loss of 81 per cent. in French publications; 84 per cent. in German publications and a gain of 7 per cent. in the publications of Great Britain. These figures, therefore, show an increase of 9 per cent. of French publications, and a loss of 15 per cent. of German publications over the year 1915, and an increase of 41 per cent. in the publications of Great Britain over 1915.

It must be understood, however, that these figures relate to the new publications received by this Library, and are not intended to represent an authoritative statement of the number of medical publications issued in the countries mentioned.

During the past year the Library Endowment Funds have been increased by the Morris Longstreth Library Fund, $7110.08, and an addition to the Louis A. Duhring Library Fund of $4656.70.

Total amount of Library Endowment Funds at this date: $305,461.88.

In closing, a comment in regard to the storage of our books and periodicals in open stacks seems wise and pertinent. The stacks for this Library were built in the most approved manner at the time of their erection (1908); fire-proof, according to modern construction; a window opposite each aisle in the stacks and a window at the ends of the main corridor on each floor; the window-panes of wire-glass set in steel frames with an automatic low-fused steel fire curtain for each window—light and air, according to the opinions of the time, being absolutely necessary for the care and preservation of books. It is impossible to fit steel casement windows tight enough to prevent the entrance of more or less dust, according to location; and the most serious problem in the use of open stacks is to prevent damage to the books from the constant handling in the attempt to keep them sufficiently free from dust, to be ready for use when called for. This building is so situated that the dust, which penetrates every closed window, including the stacks, contains a large percentage of soot, a form of dust which cannot be removed entirely with a high-power vacuum cleaner, but only by wiping with cloths, and then a smudge is left which shows, more or less, on the tops of the books. The comparison, if it could be made at present, would show a marked difference in the general condition of the books that were brought to this building after, say, twenty-five years in the locked cases at Thirteenth and Locust Streets, and after nine years in their present location in open stacks.

Modern lighting (electricity) and forced ventilation (filtered air with exhausts) in stacks built without windows or skylights, in the opinion of the Committee, furnish the only feasible plan for the proper care of large collections of books, open and ready for use at all times. What little dust is carried in and accumulates can readily be disposed of by powerful blowers in connection with an exhaust on each floor, without handling the books.

It seems urgently necessary that the present windows should be so sealed as to be absolutely dust-proof, or perhaps completely closed. In either case a special lighting and ventilating system

would have to be installed. These measures, it need hardly be added, would entail a not inconsiderable expenditure of money. There can be no doubt, however, that the Library is at present threatened by a deterioration of the books upon the stacks.

Respectfully submitted,
FRANCIS X. DERCUM, M.D.,
Chairman.

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